ACUTE HEMODYNAMIC RESPONSES TO STRENGTH TESTING AND RESISTANCE EXERCISE IN PATIENTS WITH LEFT VENTRICULAR DYSFUNCTION

BY

GALILA WERBER

A DISSERTATION PRESENTED TO THE GRADUATE SCHOOL
OF THE UNIVERSITY OF FLORIDA IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

UNIVERSITY OF FLORIDA

1997

I would like to dedicate this work to
my mother, Lucy Werber, and my brother, Alex Werber,
for their love, support, and encouragement without whose help I would not have made it this far;
and in the memory of my late father, Martin Werber, my guiding light.
I love you all.

ACKNOWLEDGEMENTS

I would like to give specials thanks to Dr. Michael Pollock, Ph.D., my committee chair, for his support, encouragement and understanding during my graduate career at the University of Florida. His guidance in the completion of this dissertation is deeply appreciated. I am also grateful for employment as a research assistant under his guidance.

I extend thanks to Dr. David Lowenthal, M.D., Ph.D., for serving on my committee and for his support, reassurance and patience throughout my doctoral graduate work. Furthermore, I am also grateful to him for the opportunity for employment as a research assistant.

I would like to acknowledge Dr. Philip Posner, Ph.D., for serving on my committee. His dedication to research and to all students and his kindness have greatly influenced my graduate pursuits.

I would also like to thank Dr. Randy Braith, Ph.D., for serving on my committee. His knowledge and experience in cardiac rehabilitation have provided me with invaluable insight.

In addition, I would like to give special thanks to Dr. Keith Tennant, Ph.D., and the College of Health and Human Performance at the University of Florida for employment as a graduate assistant in the Department of Exercise and Sport Sciences as an activity instructor in the Sport and Fitness Classes.

I would like to acknowledge Dr. William Brechue, Ph.D., for his assistance in the design of this project.

I extend thanks to Dr. Michael Sagiv, Ph.D., for employment as a lecturer in the Zinman College of Physical Education at Wingate Institute, Israel, and for providing the facility and equipment for data collections for this dissertation.

I would like to thank Anat Shaar, Noga Fisher and Hinda Annenburg for their support and help in data collection. I would like to give special thanks to Dr. Ehud Goldhammer, M.D., for analyzing the echocardiographic data. I wish to acknowledge Michal Arnon, M.Ed., and Aviva Zeev, M.S., for their invaluable help in data analysis.

I would like to thank Dr. David and Mrs. Orva Kaufmann for being my second family. I give special thanks to Dr. Robert Cade, M.D., for his kindness and financial support.

I would like the acknowledge the secretaries at the Center for Exercise Science and my subjects who dedicated their efforts to this dissertation.

Finally, I would like to thank my friends across both ends of the Atlantic ocean, Orit Israel, Maritte Witz, Katty Dayan, Linda Garazarella Deborah Herring and Diego deHoyos, who helped me through difficult times and give the true meaning to the word friendship.

TABLE OF CONTENTS

pages

ACKNOWLEDGMENTS.. iii

LIST OF TABLES.. viii

LIST OF FIGURES.. x

ABSTRACT.. xii

CHAPTERS

1. INTRODUCTION.. 1

Purpose of the Study.. 6
Hypotheses.. 7
Definition of Terms.. 7
Assumption.. 9
Limitations.. 9
Significance.. 10

2. REVIEW OF LITERATURE.. 11

Pathophysiology of Left Ventricular Dysfunction.. 11
Vascular Dysfunction and Blood Flow in LVD Patients.. 13
Alteration of Skeletal Muscle in LVD Patients.. 14
Effect of Aerobic Exercise Training in LVD Patients.. 16
Hemodynamic Responses to Dynamic and Static Exercise.. 18
Hemodynamic Responses to Static-Dynamic Exercise.. 21
Hemodynamic Responses to Resistance Exercise in Cardiac Patients.. 23
Safety of Resistance Training in Cardiac Rehabilitation Programs.. 28
Health Benefits of Resistance Training for Cardiac Patients.. 33
Effects of Resistance Training on Muscular Strength.. 36
Effects of Resistance Training on Aerobic Performance.. 37
Patient Screening and Consideration.. 39
Exercise Guidelines.. 41

Summary.. 45

3. METHODS.. 48

Subjects Characteristics.. 48
Study Design.. 50
Visit 1: Experimental Protocol.. 50
    Body Composition.. 51
    Echocardiographic Measurements.. 52
    Diagnostic Graded Exercise Test.. 54
Visit 2: Experimental Protocol.. 55
Visit 3: Experimental Protocol - Maximal Strength Evaluation (Experiment 1).. 56
Visit 4: Experimental Protocol -Resistance Exercise Training Evaluation (Experiment 2).. 58
Visit 5: Experimental Protocol.. 59
Data Analysis.. 59

4. RESULTS.. 61

Descriptive Characteristics.. 61
Clinical Symptoms.. 62
Responses During Symptom Limited Exercise Test and Strength Tests.. 63
        Hemodynamic Responses during Exercise Test.. 63
        Echocardiographic Evaluations During Exercise Tests.. 65
Responses During Resistance Exercise Bouts.. 66
    Responses During Knee Extension Resistance Exercise.. 67
    Responses During One-Arm Biceps Curl Resistance Exercise.. 69
    Echocardiographic Evaluation During Knee Extension Resistance Exercise.. 72
    Echocardiographic Evaluation During One-Arm Biceps Curl Resistance Exercise.. 77
Wall Motion Abnormalities.. 79
Correlation Between Visit 4 and Visit 5.. 80

5. DISCUSSION.. 115

Responses During Strength Testing.. 115
    Safety of One-Repetition Maximum Test.. 116
    Hemodynamic Responses During Strength Test.. 118
    Left Ventricular Function During Strength Testing.. 120
Responses During Resistance Exercise.. 123
    Safety of Submaximal Resistance Exercise.. 123

Hemodynamic Responses During Resistance Exercise........................... 126
Left Ventricular Function During Resistance Exercise........................... 132
Exercise-Induced Wall Motion Abnormalities.. 135
Summary.. 137
Conclusions.. 140
Implication For Future Research.. 141

APPENDICES

A. 24-HOUR HISTORY.. 144

B. INFORMED CONSENT FORM.. 147

C. ECHOCARDIOGRAPHIC IMAGES.. 157

REFEERENCES.. 161

BIBLIOGRAPHIC SKETCH.. 176

LIST OF TABLES

Table pages

4-1 Descriptive data of the study participants (mean ± SD)........ 82

4-2 Clinical characteristics of the subjects........................... 83

4-3 Heart rate and blood pressure values at rest, peak strength and treadmill symptom-limited exercise testing (mean ± SD).. 84

4-4 Comparison between peak mean arterial pressure and total peripheral resistance values at rest, peak strength and graded exercise testing (mean ± SD)............................ 85

4-5 Left ventricular end-diastolic and systolic dimensions and volumes at rest, peak strength and treadmill symptom-limited graded exercise testing (mean ± S.D).................... 86

4-6 Ejection fraction, stroke volume, cardiac output and systolic blood pressure to left ventricular-end systolic volume ratio valued at rest, peak strength and graded exercise testing (mean ± SD).................................... 87

4-7 Work loads and rating of perceived exertion for the different intensity bouts (mean ± SD)................................ 88

4-8 Heart rate and blood pressure responses during knee extension resistance exercise (mean ± SD)........................ 89

4-9 Mean arterial pressure and total peripheral resistance responses during knee extension resistance exercise (mean ± SD).. 90

4-10 Heart rate and blood pressure responses during one-arm biceps curl resistance exercise (mean ± SD)................. 91

4-11 Mean arterial pressure and total peripheral resistance responses during one-arm biceps curl resistance exercise (mean ± SD).. 92

4-12 Changes in left ventricular end diastolic and systolic dimensions and volumes during knee extension resistance exercise (mean ± SD)................................... 93

4-13 Changes in left ventricular end diastolic and systolic dimensions and volumes during one-arm biceps curl resistance exercise (mean ± SD)................................... 94

4-14 Changes in ejection fraction, stroke volume, cardiac output and systolic blood pressure to left ventricular-end systolic volume ratio during knee extension resistance exercise (mean ± SD)... 95

4-15 Changes in ejection fraction, stroke volume, cardiac output and systolic blood pressure to left ventricular-end systolic volume ratio during one-arm biceps curl resistance exercise (mean ± SD)..................................... 96

4-16 Prevalence of wall motion abnormalities induced by exercise.. 97

4-17 Correlation values of Visit 4 and Visit 5 hemodynamic responses and echocardiographic variables during knee extension resistance exercise (n=11).............................. 98

4-18 Correlation values of Visit 4 and Visit 5 hemodynamic responses and echocardiographic variables during one-arm biceps curl resistance exercise (n=13).......................... 99

LIST OF FIGURES

Figure		pages
4-1	Peak heart rate (HR) values during symptom-limited exercise test (SL-GXT), knee extension (KE) and one-arm biceps curl (BIC) strength tests (mean ± SE)……………….	100
4-2	Peak systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) during symptom-limited exercise test (SL-GXT), knee extension (KE) and one-arm biceps curl (BIC) strength tests (mean ± SE)………………………………………	101
4-3	Heart rate (HR) response at rest, knee extension resistance exercise and recovery during 20, 40 and 60% of 1-RM (mean ± SE)………………………………………………	102
4-4	Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) responses at rest, knee extension resistance exercise and recovery during 20, 40 and 60% of 1-RM (mean ± SE)…………………………….	103
4-5	Heart rate (HR) response at rest, one-arm biceps curl resistance exercise and recovery during 20, 40 and 60% of 1-RM (mean ± SE)………………………………………	104
4-6	Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) at rest, one-arm biceps curl resistance exercise and recovery during 20, 40 and 60% of 1-RM (mean ± SE)……………………	105
4-7	Comparison of heat rate (HR) response between knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different work load levels (mean ± SE)………….	106

4-8 Comparison of systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) responses between knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different work load levels (mean ± SE).. 107

4-9 Comparison of rate pressure products (RPP) responses between knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different work load levels (mean ± SE).. 108

4-10 Comparison between peak rate pressure product (RPP) values during symptom limited exercise test (SL-GXT), knee extension (KE) and one-arm biceps curl (BIC) strength tests and resistance exercise (mean ± SE)........................ 109

4-11 Changes in left ventricular end diastolic dimension (LVEDD) and left ventricular end systolic dimension (LVESD) from rest to exercise during knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different levels of submaximal work loads (mean ± SE)........ 110

4-12 Changes in left ventricular end diastolic volume (LVEDV) and left ventricular end systolic volume (LVESV) from rest to exercise during knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different levels of submaximal work loads (mean ± SE)......................... 111

4-13 Changes in ejection fraction, stroke volume and cardiac output during knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different levels of work loads (mean ± SE)... 112

4-14 Prevalence of resting and exercise-induced wall motion abnormalities.. 113

4-15 Prevalence of resting and exercise-induced wall motion abnormalities at submaximal resistance exercise............... 114

Abstract of Dissertation Presented to the Graduate School
of the University of Florida in Partial Fulfillment of the
Requirements for the Degree of Doctor of Philosophy

ACUTE HEMODYNAMIC RESPONSES TO STRENGTH TESTING AND RESISTANCE EXERCISE IN PATIENTS WITH LEFT VENTRICULAR DYSFUNCTION

By

Galila Werber

December, 1997

Chairman: Michael L. Pollock
Major Department: Exercise and Sport Sciences

Left ventricular dysfunction (LVD) results in reduced exercise capacity and loss of skeletal muscle mass, strength, and endurance. Resistance training has been shown to offset some of these losses in low risk cardiac patients. However, there is a lack of guidelines and a reluctance to use resistance training in low-moderate risk LVD patients ($30\% \leq$ ejection fraction (EF) $\leq 49\%$) due to insufficient data concerning its safety. The present study was designed to evaluate the safety of strength testing and resistance exercise in low-moderate risk cardiac patients with LVD. Fifteen LVD patients 65±6.5 years of age were studied during rest, and exercise and recovery from a 1-repetition maximum (1-RM) test to determine maximal strength using a one-arm biceps curl (BIC) and bilateral knee extension (KE) exercise. On a separate day, patients performed 10-15 repetitions for each exercise at 20, 40 and 60%of 1-RM. Safety was defined by measures of increased signs and symptoms such as exacerbated blood pressure (BP), electrocardiographic changes, angina pectoris, arrhythmias and reduced left ventricular (LV) function using echocardiographic assessment as compared to the results from a symptom-limited graded exercise test (SL-GXT). Peak rate pressure products were lower ($p \leq 0.05$) for both KE and BIC 1-RM resistance exercise at 60% 1-RM compared to SL-GXT (146, 179 vs. 254 $\text{mmHg}\cdot\text{min}^{-1}10^{-2}$, respectively). Echocardiographic evaluation of LV function during 1-RM strength tests demonstrated a maintenance of LV function. During resistance exercises, heart rate (HR) and BP responses increased ($p \leq 0.05$) with increased work load and with increased active muscle mass (BIC to KE), however, they remained in the range of 60-85% of SL-GXT values, which is the recommended range for aerobic exercise prescription for cardiac patients. Left ventricular function demonstrated a slight increase during both resistance exercises by echocardiographic means. There was a small but significant decrease in EF values during 60% 1-RM of KE exercise compared to rest (40 vs. 42%, respectively). Increases in new wall motion abnormalities were similar for SL-GXT and 1-RM testing (~5%). Knee extension and BIC exercises at 60% 1-RM showed only a 7.6% and 5.7% increase in new wall motion abnormalities, compared to SL-GXT; but there were no differences during exercise at 20 and 40% of 1-RM. There were no adverse effects on LV contractility as suggested by SBP/LV end systolic volume ratio (2.1 during KE 60% 1-RM vs. 1.5 at rest). The findings of this study suggest that 1-RM strength testing and resistance exercise (10-15 repetitions) using the KE and BIC exercises at 20, 40 and 60% of 1-RM are safe for patients with low-moderate LVD.

CHAPTER 1
INTRODUCTION

Cardiac rehabilitation is a primary treatment modality for patients who have cardiovascular related diseases. The principal goal of these programs is to restore physical, psychological and vocational function in cardiac patients. Traditionally, cardiac rehabilitation programs have mainly emphasized lower extremity aerobic exercise (i.e., walking, stationary cycling, stair climbing, etc.) (Goldberg, 1989; Pollock and Wilmore, 1990). Resistance exercise was not endorsed since it had been regarded as hemodynamically hazardous for patients with cardiovascular disease or with high risk factors for a future cardiac event. The primary concern was that resistance training might cause an excessive burden on the myocardium due to exaggerated blood pressure (BP) responses, which in turn, could cause higher rate pressure products leading to more ischemic events and arrhythmias (Atkins et al., 1971; Barnard et al., 1973; Jackson et al., 1973; Keul et al., 1981; Mullins and Bloqmvist 1973).

Various vocational, recreational and daily living activities such as carrying groceries, luggage, or doing yard work place demands on the cardiovascular system which more closely resemble heavy resistance exercise than aerobic exercise. Moreover, many cardiac patients lack the physical strength or mental confidence to perform these common daily tasks (Butler et al., 1987; Faigenbaum et al., 1990; Franklin et al., 1991). Therefore, it is important to recognize that cardiac patients require a minimum threshold level of strength for performing daily living activities, equal to those of a healthy individuals (Sparling and Cantwell, 1989).

During the last two decades ample evidence has been accumulating suggesting that resistance exercise training in cardiac patients may be less hazardous than was once presumed, especially in low-moderate risk patients (DeBusk et al., 1978; DeBusk et al., 1979; Franklin et al., 1986; Franklin et al., 1991; Kerber et al., 1975; McKelvie et al., 1995; Saldivar et al., 1983; Stewart et al.,1988; Verrill and Ribisl, 1996). The benefits of resistance training for healthy individuals and for cardiac patients includes improved muscular strength and endurance, bone mineral density, muscle mass, functional capacity, metabolism, and improved self-image and self-confidence (Ewart, 1989; Kelemen et al., 1986; Sparling et al., 1990; Stone, 1988). Increased muscular strength resulting from resistance training allows a submaximal work load to require a relatively lower effort and consequently is perceived as less of a strain. Furthermore, the enhanced strength can lessen the likelihood of musculoskeletal injuries which often accompany physical activity. Consequently, patients who resistance train will be able to perform strenuous daily activities with lesser percentage of maximal strength, a diminished perception of effort and decreased risk of injuries, resulting in increased functional capacity, independent life style and enhanced quality of life (McCartney et al., 1991; Stewart, 1989; Stone et al., 1991).

Resistance training can produce a small increase in aerobic capacity, which is associated with the increase in strength and muscle mass (Gettman and Pollock, 1981; Hickson et al., 1980). Exercise capacity of patients with cardiac disease can be limited by leg fatigue resulting in the termination of exercise without coexisting evidence of cardiorespiratroy limitation. Studies conducted in cardiac rehabilitation programs have demonstrated enhanced treadmill performance in patients who participated in circuit weight training. Such alterations in performance were not observed in the control group (Hung et al., 1984; Kelemen et al., 1986; McCartney et al., 1989; McCartney et al., 1991; Oldridge et al., 1989). In cardiac patients who are severely deconditioned, resistance exercise can cause muscular changes that can lead to enhanced ability to engage in aerobic training, thus improving aerobic capacity.

Patients with left ventricular dysfunction (LVD) display varied chronic responses such as reduced cardiac outputs, compensatory neuroendocrine responses, reduced exercise capacity with symptoms of dyspnea and fatigue which leads to physical inactivity, skeletal muscle atrophy and muscle weakness (Curtiss et al., 1978; Drexler et al., 1988; Drexler et al., 1992; Mancini et al., 1992; Smith et al., 1993; Zelis et al., 1988). Skeletal muscle alterations, which are found in LVD patients, are similar to those observed with prolonged deconditioning or immobilization and are related to the duration of myocardial dysfunction. Therefore, improve the patient's exercise capacity by including exercise training into their program will help reverse this abnormal process (Drexler et al., 1992; Mancini et al., 1989; Mancini et al., 1992). The beneficial effects of aerobic exercise in LVD patients, have been well documented. The results of these studies demonstrated increased aerobic capacity through peripheral adaptation (Hanson,1994; Stratton et al., 1994; Sullivan et al., 1989). Symptoms such as tiredness, dyspnea with exertion, and overall weakness are most common in LVD patients. Thus, engaging in resistance training may result in muscular changes that can lead to improvements in muscle strength and endurance and aerobic performance via increasing muscle mass and strength (Hanson, 1994; Massie et al., 1988; McCartney et al., 1989; Wilson et al., 1985).

During the late 80s and early 90s, the conventional inclusion criteria of cardiac patients for a resistance training program were mainly directed toward low risk patients who were already participating in a traditional aerobic exercise program for at least 3 months (Franklin et al., 1991; Kelemen, 1989; McKelvie and McCartney, 1990; Sparling and Cantwell, 1989). Generally, exclusion criteria for resistance training resembled those used for any outpatient cardiac rehabilitation program. (Franklin et al., 1991; Sparling and Cantwell, 1989). Patients were excluded for the following reasons: unstable angina, uncontrolled hypertension (systolic BP (SBP) > 160 mmHg or diastolic BP >100 mmHg), uncontrolled arrhythmias, a recent history of congestive heart failure, a maximal aerobic capacity of less than 6-7 metabolic equivalents (METs) (1 MET = 3.5 $ml \cdot kg^{-1} \cdot min^{-1}$) during symptom limited graded exercise test (SL-GXT), or poor LV function (ejection fraction (EF) < 45%) (Franklin et al., 1991; Kelemen, 1989; Sparling and Cantwell, 1989; Verrill et al., 1992). However, recent studies performed in early outpatient cardiac rehabilitation settings (phase II) as soon as 2 weeks after acute myocardial infarction demonstrated no adverse cardiovascular responses in properly selected patients participating in resistance training at 40% of maximal voluntary contraction (MVC) (Daub et al., 1996; Squires et al., 1991; Stewart et al., 1995). In light of these findings the revised Exercise Standards of the American Heart Association (AHA) (AHA, 1995), American Association of Cardiovascular and Pulmonary Rehabilitation (AACVPR)

(AACVPR, 1995) and recent American College of Sports Medicine (ACSM) (ACSM, 1995) guidelines for exercise testing and prescription include less conservative contra-indications for resistance exercise training for cardiac outpatients. Low-moderate risk patients who in the past were excluded from the resistance training regimen are considered now as candidates who can exercise safely with weight using a lighter load, for example., 20% of MVC. Such patients include older cardiac patients, patients with LVD (EF>35%), patients with mitral valve prolapse syndrome and heart transplant patients (Braith et al., 1993; Braith et al., 1994; Braith et al., 1996; Daub et al., 1996; Frederickson, 1988; McKelvie et al., 1995; Munnings, 1993; Verrill and Ribisl, 1996). The current AACVPR, ACSM and AHA recommendations for resistance training for low-moderate risk cardiac patients consists of 8-10 exercises which train the major muscle groups of the body, one set of 10-15 repetitions at a load of 30%-50% of the one-repetition maximum (1-RM) for each exercise, performed 2-3 days per week (AACVPR, 1995; ACSM, 1995; AHA, 1995). Once 15 repetitions can be comfortably completed by the patient the load can be raised by an additional 5% (ACSM, 1995; AHA, 1995; Sparling and Cantwell, 1989).

The AACVPR, AHA and ACSM guidelines for resistance exercise in low risk cardiac patients are based on the guidelines previously developed for healthy adults. However, training intensity for cardiac patients is lower (moderate fatigue vs. maximal effort), and the number of repetitions is higher (10-15 vs. 8-12) than is recommended for healthy adults. Most of previously published studies concerning resistance training in cardiac patients investigated safety of resistance training in low risk patients. However, there are no specific guidelines for strength testing or resistance exercise training for low-moderate risk cardiac patients with LVD due to insufficient data on safety. Therefore, the purpose of the present study was to determine the effects of 1-RM strength testing and resistance exercise (10-15 repetitions at 20, 40 and 60% of 1-RM) in cardiac patients with moderate LVD (30% ≤ EF ≤ 49%). It is hoped that this information can be used in helping to prescribe safe resistance training programs in low-moderate risk cardiac patients.

Purpose of the Study

The present study was designed to appraise the safety of strength testing and resistance exercise in low-moderate risk cardiac patients with LVD (30% ≤ EF ≤ 49%). Two specific aims were proposed:

1. to establish the safety of strength testing (1-RM), and
2. to establish the safety of repetitive resistance exercise at various submaximal intensities using 10-15 repetitions at 20, 40 and 60% of 1-RM.

Safety was defined by measures of signs and symptoms such as exacerbated BP (auscultation), angina pectoris, electrocardiographic (ECG) changes (ST segment depression ≥2 mm), arrhythmias and reduced LV function using echocardiographic evaluation.

Hypotheses

The following hypotheses concerning the safety of strength testing and resistance exercise were proposed:

1. the performance a of 1-RM test is safe and does not impose any apparent additional risks on cardiac patients with LVD,
2. there are no significant differences in safety between resistance exercise bouts of 10 to 15 repetitions at 20, 40 and 60% submaximal work loads based on the 1-RM test than for SL-GXT, and
3. there are no significant safety differences between arm resistance exercise vs. leg resistance exercise.

Definition of Terms

Low risk patients include cardiac patients with no significant LV dysfunction (EF≥ 50%) and no resting or exercise-induced ischemia or arrhythmias; status post uncomplicated myocardial infarction, coronary artery bypass graft (CABG), angioplasty, or arthrectomy; and with functional capacity above 6 METs 3 weeks after a cardiac event.

Moderate risk patients include cardiac patients with mild to moderately depressed LV function (30 ≤EF≤ 49%); exercise-induced myocardial ischemia; and functional capacity ≤ 5-6 METs 3 weeks or more after a cardiac event.

High risk patients include cardiac patients with severely depressed LV function (EF≤ 30%); complex ventricular arrhytmias at rest, or appearing or increasing with exercise; marked exercise-induced myocardial ischemia; exertional hypotension (≥ 15 mmHg decrease in SBP during exercise); low functional capacity; myocardial infarction complicated by chronic heart failure, cardiogenic shock and/or complex ventricular arrhythmias; and survivor of cardiac arrest.

Hemodynamics is the study of blood flow regulation in the vascular beds, involves the interrelationship between pressure, flow and resistance.

Ejection fraction (EF) is the percent of left ventricular diastolic volume that is ejected during systole.

$$EF = \frac{\text{end diastolic volume - end systolic volume}}{\text{end diastolic volume}}$$

Wall motion is the movement of the left ventricle wall during systole. The assessment of wall motion is performed by dividing the ventricle wall into regions which are being scored in respect to their movement.

Isometric exercise is a muscle contraction performed against a fixed resistance, where tension is developed without change in range of motion.

Isotonic exercise is a muscle contraction against resistance, the load remains constant, with the resistance varying with the angle of the joint throughout full range of motion, for example, lifting free weights.

One repetition maximum (1-RM) is the maximal amount of weight that can be lifted during one dynamic repetition throughout full range of motion using a good form and technique.

Resistance exercise is the method for developing muscle strength and endurance by having the muscle contract against an opposing load (resistance). This is accomplished by performing a certain number of repetitions of weightlifting through a full range of motion at varying levels of intensity, e.g. 20, 40 or 60% of 1-RM.

Assumption

It is assumed that all patients followed all instructions and provided their best effort during the SL-GXT and 1-RM strength tests.

Limitations

1. Continuous, intraarterial pressure measurement is the most accurate and reliable method for measuring BP. However, due to inherent risk of arterial catheterization in LVD patients, indirect measurements of BP were utilized. While it has been reported that resting SBP determined by auscultation is on average 13% lower than the values obtained simultaneously from a brachial catheter (Wiecek et al., 1990), comparative values demonstrate a high correlation (Sagiv et al.,1995). As for diastolic BP it was found to be similar using either technique. The error in SBP associated with the different techniques is constant and maintained during and after either arm or leg exercise. In order to better approximate and maximize measurement accuracy of BP during the lifting phase, auscultations were performed at the mid point of each set and towards the final repetitions of each intensity of exercise, rather than after the exercise. While absolute values for SBP may be underestimated, indirect BP measurements should give an accurate measure of the differences observed in BP during the actual lift.

2. Measurements of BP during 1-RM were not taken during the lift but performed only immediate post exercise due to the short time span of the procedure. In order to minimize the error between the actual values attained during the lift phase and the reading at the end of the lift, the cuff was inflated prior to the lift of the weight.
3. The estimation of cardiac output from volume measures observed from the echocardiographic images has a moderately large standard error (approximately ± 2 $L \cdot min^{-1}$ (Perez-Gonzales et al. 1981)). However, this was minimized because the same technician collected the data and the same cardiologist analyzed it. Thus, even though absolute volume may be under/overestimated, the change in volume should be accurately reflected.
4. Only knee extension and one-arm biceps curl strength test and resistance exercises were evaluated.

Significance

1. There are no specific guidelines for strength testing or resistance exercise training for low-moderate risk cardiac patients with LVD due to insufficient data on safety. Thus, the information obtained from this study will further the understanding of the cardiovascular responses to strength testing and low to moderate intensities of resistive exercise in LVD patients ($30\% \le EF \le 49\%$).
2. If the designed proposal as suggested is found effective and positive, it could help facilitate the weightlifting training guidelines for cardiac patients with LVD.

CHAPTER 2
REVIEW OF LITERATURE

The following review will cover three major topics concerning resistance training and left ventricular dysfunction patients. The first part of the review will discuss the pathology and manifestations of reduced left ventricular function. The second part will cover the hemodynamic aspect of resistance exercise in healthy individuals and in cardiac patients. The last portion will review the contemporary literature regarding the safety and efficacy of resistance training in low risk cardiac patients, including the current trends in exercise guidelines for this patients population.

Pathophysiology of Left Ventricular Dysfunction

Heart failure is defined as the pathological state in which the heart is unable to pump blood at a rate corresponding to the body's metabolic demands. The most common etiologies for the reduced myocardial function are extensive myocardial infarction as a result of coronary artery disease (CAD) and idiopathic dilated cardiomyopathy. Other causes such as valvular and congenital heart disease, hypertension, drug toxicity, coronary emboli and myocardial trauma can also play a significant role in left ventricular dysfunction (LVD) (Blumenfeld and Laragh, 1994; Codd, 1989; Fozzard et al., 1991; Francis and Cohn, 1990).

Changes in the mass, volume, and shape of the left ventricle seems to be critical for the development of the heart failure syndrome once myocardial dysfunction is present (Blumenfeld and Laragh, 1994; Francis and Cohn, 1990). The increase in chamber size seen in heart failure patients results in higher wall stress and increased energy demand which leads to progressive myocyte necrosis, fibrosis, and further chamber dilation (Hanson,1994; Treasure and Alexander, 1993; Vatner and Huttinger, 1993; Weber et al., 1985). The primary abnormalities of the ventricular pump performance are impaired diastolic filling or systolic emptying, which leads to a reduction in left ventricular ejection fraction (EF) and resulting in an impaired cardiac output (CO) and tissue oxygenation (Fozzard et al., 1991).

Survival depends on the perfusion pressure of vital organs (Fozzard et al., 1991). Thus, in order to prevent a fall in blood pressure (BP), due to the reduced left ventricular (LV) function, compensatory mechanisms must be employed. Consequently, heart failure is a complex manifestation of chronic responses involving the impaired cardiac function, autonomic nervous system, endocrine organs, skeletal muscle, kidneys and regional vascular beds resulting in clinical symptoms of dyspnea and fatigue (Fozzard et al., 1991; Francis and Cohn, 1990; Hanson, 1994; Smith et al., 1993). Reduced renal sodium and water excretion which leads to volume overload, elevation of sympathetic nervous system response resulting in an increased plasma norepinephrine levels, and increased plasma renin activity are common characteristics of the heart failure syndrome (Bayliss et al., 1985; Curtiss et al., 1978; Francis and Cohn,1990; Just, 1991; Levine et al., 1982).

Vascular Dysfunction and Blood Flow in LVD Patients

Due to reduced cardiac function, some circulatory compensatory mechanisms are used in order to maintain blood supply and perfusion pressure. In acute heart failure, sympathetic peripheral vasoconstriction, with increased chronotropic and inotropic responses, is designed to restore circulatory homeostasis. In addition, vascular constriction is mediated via the renin-angiotensin-vasopressin system, which is being activated in proportion to the severity of heart failure (Ryden, 1988; Weber et al., 1985; Zelis et al., 1988). Impairment of the endothelium-mediated flow-dependent vasodilation may also be responsible for the reduced arterial compliance in chronic congestive heart failure, by limiting blood flow (BF) to the working organ and maintaining increased afterload for the diseased ventricle (Just, 1991; Drexler et al., 1988).

The maximal exercise capacity of patients with LVD is frequently reduced (Drexler et al., 1987; Musch and Terrell, 1992; Wilson et al., 1984). This reduction in exercise performance is often associated with decreased skeletal muscle BF responses to a given work load (Drexler et al., 1987; Wilson et al., 1984). Wilson and associates (1984) studied whether maximal exercise capacity in patients with LVD correlates with the sufficiency of BF to the working skeletal muscle. The investigators used leg BF, oxygen extraction, and venous lactate concentration as indices to assess nutritional flow to skeletal muscle during maximal cycle exercise. Their results demonstrated impaired BF to skeletal muscle, with a correlation between the severity of exercise intolerance and the degree of impairment of nutrient BF to the working muscle. Musch and Terrell (1992) found a greater deficit in BF to the working muscle in rat hindlimb, as the size of the myocardial infarction and the amount of left ventricular dysfunction increased.

Alteration of Skeletal Muscle in LVD Patients

Exertional fatigue is the major limiting symptom in heart failure patients. Poor correlation has been found between exercise performance and state of the reduced left ventricular function. Moreover, increased CO during exercise, exerted by pharmacological intervention, failed to increase exercise capacity and peak oxygen consumption in heart failure patients (Adamopoulos and Coats, 1991; Drexler et al., 1988; Drexler et al., 1992; Massie, et al., 1988; Wilson et al., 1984; Wilson et al., 1985). Thus, intrinsic skeletal muscle abnormalities may also play an important role for the reduced exercise tolerance in patients with chronic heart failure. Studies with ^{31}P nuclear magnetic resonance in heart failure patients and healthy subjects have demonstrated a progressive rise in inorganic phosphorus to phosphocreatine (Pi/Pcr) ratio as oxygen consumption increased during exercise in both groups. However, heart failure patients demonstrated a steeper slope of Pi/Pcr ratio compared to the healthy subjects. Accordingly, heart failure patients depleted muscle Pcr more rapidly and at lower a workload compared to healthy adults, which might be a characteristic of impaired oxidative phosphorylation in the exercising skeletal muscle (Adamopoulos and Coats, 1991; Mancini et al., 1989; Mancini et al., 1992; Pierre-Yves et al., 1990; Rajagopalan et al., 1988; Wiener et al., 1986; Wilson et al., 1985).

Lower pH values, with early onset and increased glycolytic metabolism were documented in heart failure patients at lower work loads compared to control subjects

(Adamopoulos and Coats, 1991; Drexler, 1991; Mancini et al., 1989; Mancini et al., 1992; Massie et al., 1987; Massie et al., 1988; Pierre-Yves et al., 1990; Wiener et al., 1986; Wilson et al., 1985). Massie and colleague (1988) demonstrated that during low intensity work load, e.g. 33% of peak maximum work load, subjects with chronic heart failure exhibit significantly lower pH and higher Pi/Pcr ratios, which indicates an earlier and higher rate of glycolytic metabolism. Biochemical analysis demonstrated reduced mitochondrial enzyme concentrations, such as succinate dehydrogenase, citrate synthetase, and 3-hydroxyacyl-CoA-dehydrogenase, in heart failure patients compared to normals (Drexler et al., 1991; Drexler et al., 1992; Drexler et al., 1988; Lipkin, 1988; Mancini et al., 1992; Sullivan et al., 1989). Drexler and associates (1992) showed a reduction of 20% in mitochondrial volume density, and surface density of mitochondrial cristae in chronic heart failure patients. The investigators also found a significant decrease in cytochrome oxidase activity, which indicates reduced oxidative capacity of the working muscles.

Patients with heart failure exhibit muscle fiber type alterations, such as a shift in fiber type distribution, fiber atrophy, reduced skeletal muscle capillary density, and decreased capillary to muscle fiber ratio (Adamopoulos and Coats, 1991; Caforio et al., 1989; Drexler et al., 1988; Drexler et al., 1992; Yancy et al., 1989). Sabbah et al. (1993), induced heart failure in 17 dogs, for a period of 3-4 months by sequential intracoronary microembolism. The progressive decline in left ventricular function was accompanied by a progressive decrease in the proportion of type I fibers and a progressive increase in the proportion of type II fibers. In addition, cross sectional area of both fiber types decreased gradually during the developing heart failure with no preferential atrophy of either muscle fiber types. Mancini et al. (1992) obtained muscle biopsies from the gastrocnemius muscle of 22 heart failure patients, the results showed a shift in fiber type distribution with a reduction in type I and type IIa fibers and a significant 33% increase in the proportion of type IIb fibers. Since IIb type have less oxidative capacity than type I and IIa fibers, the reduced aerobic tolerance in heart failure patients could be attributed partly to the shift in the ratio of skeletal muscle type I to type II fibers (Drexler et al., 1992; Sabbah et al., 1993; Yancy et al., 1989). Muscle atrophy seen in patients with heart failure, may contribute to their exercise intolerance and muscle metabolic abnormalities. Jondeau et al. (1992), demonstrated an increase in peak oxygen consumption in severe chronic heart failure patients during exercise while combining both upper and lower limb exercise, compared to lower limb exercise alone.

Effect of Aerobic Exercise Training in LVD Patients

The skeletal muscle alterations seen in heart failure patients are similar to those observed with prolonged deconditioning or immobilization and are related to the duration of the reduced myocardium function (Drexler et al., 1992). Since these muscle alterations contribute to exertional fatigue, improving the exercise capacity of LVD patients, by aerobic exercise training, may reverse this abnormal process (Drexler et al., 1992; Mancini et al., 1989; Mancini et al., 1992).

The biochemical and histological changes demonstrated in heart failure patients resemble those that occur due to training cessation. Endurance exercise training induces adaptations in skeletal muscle, such as increased mitochondrial volume and mitochondrial content, and increased capillary supply. This is accompanied by metabolic changes, such as slower utilization of glycogen, a greater reliance on fat oxidation, and less lactate production during exercise at a given work load (Drexler et al., 1992; Sullivan et al., 1989). Therefore, aerobic exercise training appears to help reverse the intrinsic muscle alteration and enhance exercise tolerance in heart failure patients.

Endurance exercise training in LVD patients results in lower resting and submaximal heart rates at standard relative work loads. Furthermore, submaximal, as well as, maximal exercise performance increases due to training (Stratton et al., 1994; Sullivan et al., 1989). Significant decreases and delays in blood lactate accumulation, during submaximal exercise coupled with increased peak lactate production, due to improved functional capacity, were documented in heart failure patients who engaged in endurance training (Hanson, 1994; Sullivan et al., 1989). Sullivan et al. (1989) demonstrated that blood lactate levels at submaximal exercise were reduced without improvements in CO. Thus, peripheral metabolism is important in determining the onset of lactate production and appears to be independent of central hemodynamics. The impaired oxidative capacity of skeletal muscle in heart failure patients can be improved by endurance exercise training (Adamopoulos and Coats, 1991; Drexler et al., 1992, Hanson, 1994; Stratton et al., 1994). Stratton et al. (1994) demonstrated increased rate of Pcr resynthesis, increased maximal rate of mitochondrial ATP synthesis, and higher submaximal levels of pH with increased duration of endurance exercise, following one month of forearm exercise.

In summary, insufficient active muscle mass and intrinsic alterations in skeletal muscle metabolism can act as predominant limiting factors for exercise intolerance in patients with reduced myocardial function. Therefore, the functional capacity of patients with LVD is limited not only by the capacity of the oxygen transport system, but also by the oxidative capacity of the working muscle. Nevertheless, part of the abnormal changes in skeletal muscle in this patient population may be reversed as a result of aerobic exercise training.

Hemodynamic Responses to Dynamic and Static Exercise

Muscular activity is associated with changes in cardiovascular function leading to an increase in BF through the active muscles. Static (isometric) and dynamic (isotonic) exercise produce different metabolic, hormonal and cardiovascular responses. Therefore, the mode of muscle contraction (dynamic or static) is a specific determinant of the cardiovascular response (Asmussen, 1981; Blomqvist and Saltin, 1983; Crawford et al., 1979; Keul et al., 1981).

Dynamic or rhythmic muscle activity causes large increases in CO and heart rate (HR), while mean arterial pressure (MAP) changes very little. Generally, systolic blood pressure (SBP) increases with an increase in the workload, closely mimicking changes in CO, while diastolic pressure remains unchanged or is slightly decreased (Blomqvist et al., 1981; Crawford et al., 1979; Keul et al., 1981). The increased muscle activity generates an enhanced metabolic demand, which is met by a local response of vasodilation resulting in an increase in muscle BF. Thus, during dynamic exercise peripheral resistance decreases as a result of dilation of the vascular bed in the active muscles (Perez-Gonzalez, 1981). The extent to which HR, CO, and SBP increase during dynamic exercise depends on the muscle group being used and the intensity of work performed (Blomqvist and Saltin, 1983; Clausen, 1977).

Acute circulatory adaptation to static exercise is regulated by both central and peripheral mechanisms (Asmussen, 1981; Helfant et al., 1971; Perez-Gonzalea, 1981; Seals et al., 1983). The central mechanism involves the irradiation of impulses from the motor cortex to the medullary cardiovascular center. It is associated with an abrupt pressor response, a significant increase in SBP, DBP, and MAP resulting in an intense afterload on the left ventricle (LV), coupled with an augmented HR and CO response (Asmussen, 1981; Helfant et al., 1971; Perez-Gonzalea, 1981; Seals et al., 1983). The pressor response serves to increase the perfusion pressure in the active muscles, in which BF is impeded by muscular mechanical compression (Helfant et al., 1971; MacDougall et al., 1985). The peripheral mechanism consists of a reflex pathway originating in the contracting muscle. Release of metabolites from the active muscles and/or increase in the osmolarity of the interstitial fluid can activate nerve endings, which in turn provide feedback to the medullary cardiovascular center (Misner et al., 1990; Seals et al., 1983).

During isometric exercise the rise in BP and HR depend on the duration, intensity (percent of maximal voluntary contraction (MVC)), and the total of active muscle mass involved (Blomqvist et al., 1981; Lewis et al., 1983; Mitchell et al., 1980; Perez-Gonzalez, 1981; Seals et al., 1983; Tesch et al., 1988). Peripheral resistance increases as a consequence of the mechanical compression of the blood vessels due to increased intramuscular pressure, which is proportional to the MVC (Helfant et al., 1971; MacDougall et al., 1985). During maximal static contractions the BF may be impeded or even completely blocked (Asmussen, 1981; Keul et al., 1981). Thus, to overcome the increased resistance of the vascular bed (afterload), the heart must increase contractility and HR in order to maintain an appropriate CO. Echocardiographic studies have demonstrated that in spite of the expected increase in BP, HR, and CO, ejection indices did not change significantly in both normal (Crawford et al., 1979; Keul et al., 1981; Laired et al., 1979; Perez-Gonzales et al., 1981; Stefadouros et al., 1974) and cardiac patients (Kivowitz et al., 1971; Sagiv et al., 1985), suggesting an enhanced cardiac contractility.

Echocardiographic studies during submaximal dynamic exercise have shown increases in end-diastolic volume (EDV) and LV shortening velocity, with decreased end-systolic volume (ESV). This produces a more complete systolic ejection resulting in an increased stroke volume (SV) (Crawford et al., 1979; Effron, 1989; Keul et al., 1981). However, The hemodynamic responses to submaximal static exercise vary from those observed during dynamic exercise. Studies have shown a decrease or no change in LV shortening velocity and small increase in ESV, where EDV and SV did not change (Crawford et al., 1979; Effron, 1989; Keul et al., 1981). The latter resulted in a pressure overload as opposed to the volume overload which was associated with dynamic exercise.

Hemodynamic Responses to Static-Dynamic Exercise

Hemodynamic responses during resistance training exercise (weightlifting) differ from those observed during static exercise. Weightlifting consists of three different phases: the concentric contraction phase, the lockout phase, where the joint is almost fully extended, and finally, the eccentric contraction phase (Lentini et al., 1993; MacDougall et al., 1985). The exercise involves both static (concentric and eccentric contractions) and dynamic (overcoming the inertia of the weight along the full range of motion) components. Each component results in a different hemodynamic response, therefore, resistance training can be described as a static-dynamic form of exercise (Lentini et al., 1993; MacDougall et al., 1985).

Direct BP measurement during weightlifting exercise demonstrates a profound elevation in both SBP and DBP with the initiation of the concentric muscle contraction. During the eccentric phase, SBP and DBP pressure are still elevated, however they are lower than those observed during the concentric phase (Lentini et al., 1993; MacDougall et al., 1985; MacDougall et al., 1992). The amount of force which can be developed during maximal concentric contraction is less than the force which can be produced during maximal eccentric contraction. Therefore, for a given absolute load, more effort will be exerted during the concentric phase, resulting in higher BP values than during the eccentric phase (Lentini et al., 1993; MacDougall et al., 1985). Lentini et al. (1993), have reported average direct BP values of 270 mmHg of SBP and 183 mmHg DBP in young healthy adults while performing the concentric phase of heavy (high intensity) leg press exercise. In contrast, during the eccentric phase, values decreased for SBP to an average of 207 mmHg and 116 mmHg for DBP. Further significant reductions in SBP and DBP were seen during the lockout phase (Lentini et al., 1993).

MacDougall and associates (1985) demonstrated progressively higher levels of BP with each subsequent repetition while performing a heavy leg press exercise. As exercise proceeded and more repetitions were performed, additional motor units were recruited with increasing involvement of accessory muscles in order to offset fatigue. This resulted in a gradual increase in active muscle mass, which in part accounted for the progressive increase in BP (Lentini et al., 1993; MacDougall et al., 1985; Misner et al., 1990). Furthermore, as subsequent repetitions were performed, there was a progressive increase in CO due to an increased HR. This also contributed to the progressive rise in BP (MacDougall et al., 1992).

The Valsalva maneuver is an integral part of heavy resistance training and responsible for a large portion of the rise in BP that occurs with this mode of exercise . The Valsalva maneuver is a forceful expiration against a closed epiglottis, resulting in increased intrathoracic pressure and thereby impeding venous return and CO. The augmentation in intrathoracic pressure is transmitted through the aorta into the arterial tree, causing an abrupt rise in BP (Ewing et al., 1976; Lentini et al., 1993; MacDougall et al., 1992; Smith and Kampine, 1990). The BP rise is cyclic and brief and returns to normal values within 5 to 15 seconds after completing the maneuver. In order to avoid the hemodynamic strain on the circulatory system due to the Valsalva maneuver, subjects should be instructed to continue breathing while performing weightlifting. However, during heavy weight lifting, the Valsalva maneuver affords a mechanical advantage by stabilizing the trunk and consequently, cannot be avoided when subjects perform maximal or near-maximal repeated contractions to failure (Lentini et al., 1993; MacDougall et al., 1985; MacDougall et al., 1992).

In summary, the increased BP seen while performing resistance training exercise is the result of the mechanical compression of blood vessels with each muscle contraction often incorporating a powerful pressor response and Valsalva maneuver. In healthy subjects, the pressor response is associated mainly with elevated BP, HR and CO, and to a lesser extent to vasoconstriction in the non-exercising vascular beds (Bezucha et al., 1982; Ewing et al., 1976; MacDougall et al., 1985; Misner et al., 1990).

Hemodynamic Responses to Resistance Exercise in Cardiac Patients

Resistance exercise has been previously regarded as hemodynamically hazardous for patients with cardiovascular disease and for those who are at high risk for a future cardiac event. The primary concern is that resistance training may cause an excessive burden on the myocardial due to an exaggerated BP response, resulting in higher myocardial oxygen demand which may lead to more ischemic events and arrhythmias (Atkins et al., 1971; Barnard et al., 1973; Jackson et al., 1973; Keul et al., 1981; Mullins and Blomqvist, 1973). Keul et al. (1981) studied the effect of static and dynamic exercise on heart volume, contractility and LV dimensions in healthy subjects and in patients with hypertension and cardiomyopathy. The researchers concluded that in patients with myocardial infarction and/or coronary insufficiency, training programs including static exercise, are not recommended due to the associated circulatory strain. Mullins and

Blomqvist (1973) documented a large increase in LV end-diastolic pressure and the occurrence of ventricular arrhythmia with isometric handgrip exercise in cardiac patients. Consequently, static or combined static-dynamic exercise has been traditionally discouraged in cardiac rehabilitation programs. Nevertheless, many recreational and vocational activities require that patients with cardiac disease perform tasks that involve lifting and straining (Franklin et al., 1991; Sparling and Cantwell, 1989). Therefore, it is important to recognize that many cardiac patients require a minimum threshold level of strength for occupational activities and activities of daily living equal to those of healthy individuals (Sparling and Cantwell, 1989).

For more than a decade, ample evidence has accumulated suggesting that resistance exercise may be less hazardous than was once presumed, especially in low risk cardiac patients with normal LV function (DeBusk et al., 1978; DeBusk et al., 1979; Froelicher et al., 1984; Haissly et al., 1974; Kerber et al., 1975; Saldivar et al., 1983; Sparling and Cantwell, 1989, Stewart et al., 1988). DeBusk and associates (1978), compared cardiovascular responses during leg ergometry exercise to those observed during static exercise (sustained contraction at 50% of maximal forearm lifting capacity) in patients seven weeks after myocardial infarction. Ischemic ST segment depression was absent during combined static-dynamic exercise, while about 25% (e.g. 10/40) of the patients demonstrated ST depression during dynamic exercise (leg ergometer).

The rate pressure product (RPP) (HR multiplied by SBP) correlates highly with myocardial oxygen consumption and coronary BF (DeBusk et al., 1978; DeBusk et al., 1979; Gobel et al., 1977; Nelson et al., 1974). In the DeBusk et al. study (1978), maximal

RPP values during static exercise were significantly lower compared to those at which ischemic ST segment depression occurred during dynamic exercise. This was due to a lower peak HR and SBP (DeBusk et al., 1978; DeBusk et al., 1979). In another study by DeBusk et al. (1979), cardiac patients performed a treadmill test while carrying a weight of 20%, 25% or 30% of maximum forearm lifting capacity (static-dynamic exercise), and a treadmill test in which no weight was carried. Data demonstrated no worsening of the ischemic response while performing static-dynamic exercise. The RPP at the onset of ischemic ST-segment depression or angina pectoris were significantly higher during the static-dynamic exercise than during the dynamic exercise alone (DeBusk et al., 1979). These data are in agreement with other studies indicating that despite the higher RPP during combined static-dynamic exercise, cardiac patients had less anginal responses than during dynamic exercise alone (Haissly et al., 1974; Kerber et al., 1975). An important point is that the increased DBP seen during static or static-dynamic exercise provides a protective effect by increasing coronary perfusion pressure. This increase in coronary perfusion pressure improves subendocardial BF, resulting in a reduction of the development of myocardial ischemia (Bertagnoli et al., 1990; Debusk et al., 1979; Kerber et al., 1975). The rate of oxygen utilization by the myocardium is the main factor which controls coronary BF. Braunwald et al. (1958) demonstrated that a rise in arterial BP resulted in an increased myocardial oxygen demand with much greater increase in coronary BF compared to the increase in BF seen by augmenting CO. Nelson et al. (1974) demonstrated a significantly greater myocardial BF during combined static-dynamic exercise coupled with a significant increase in RPP, than found during either dynamic and static exercise alone.

Studies evaluating LV function in low risk cardiac patients during isometric contractions of both small and large muscle groups demonstrated stable global LV function (Kivowitz et al., 1971; Sagiv et al., 1985). Sagiv and colleagues (1985) studied LV responses to isometric handgrip and deadlift exercise at 30% of MVC in well trained cardiac patients. Left ventricular EF showed no significant difference from resting values for either the handgrip or deadlift exercise in both healthy adults and cardiac patients. Also, during exercise rates of systolic ejection, diastolic filling and relative ESV and EDV were statistically insignificant compared to resting values in both groups. Kivowitz et al. (1971) found that while performing handgrip exercise at 25% of MVC for 5 minutes, patients classified by the New-York Heart Association (NYHA) classification as class I and class II demonstrated an increase in LV stroke work index with small or no increase in LV end diastolic pressure. The occurrence of increased BP during isometric exercise in these patients resulted from a rise in both systemic vascular resistance and CO. However, patients classified as class III had a slight decrease in LV stroke work which was associated with a larger increase in LV end diastolic pressure. Therefore, in these patients arterial pressure increased mainly via marked elevation in systemic vascular resistance (Kivowitz et al., 1971). Nevertheless, in a recent study by McKelvie et al. (1995), LV function was found to be well-maintained in patients with congestive heart failure (CHF) (EF 27 ± 2%) while performing 2 sets of 10 repetitions at 70% of one-repetition maximum (1-RM) during unilateral leg press exercise. No significant differences were found in EDV and ESV between rest and exercise, and CO increased mainly through increased HR.

As previously discussed, the Valsalva maneuver gives a mechanical advantage by stabilizing the trunk and cannot be avoided while performing maximal or near-maximal repeated contractions to failure. (Lentini et al., 1993; MacDougall et al., 1985; MacDougall et al., 1992). Pepine and Nichol (1988) demonstrated that an increase in intrathoracic pressure caused by the Valsalva maneuver alleviated acute anginal symptoms in cardiac patients. During the maneuver, after an initial increase in intrathoracic pressure, determinants of myocardial wall tension decreased almost instantaneously, which resulted in a reduction of myocardial oxygen demand.

In summary, despite the higher RPP found during combined static-dynamic exercise, cardiac patients have less anginal responses than during maximal dynamic exercise alone (Haissly et al., 1974; Kerber et al., 1981). The increased DBP seen during isometric exercise provides a protective effect by increasing coronary perfusion pressure which improves subendocardial BF which in turn reduces the development of myocardial ischemia (Bertagnoli et al., 1990; DeBusk et al., 1979; Kerber et al., 1975). Cardiac patients with normal LV function have an increased or unchanged SV stroke work index with a small rise in the cardiac index, suggesting enhanced LV function during isometric exercise. Patients with abnormal LV function demonstrate a decrease in LVEF, no change or even reduced SV stroke work index and cardiac index, with significantly increased LV end diastolic pressure during this type of exercise (Elkayam et al., 1985; Kivowitz et al., 1971; Painter and Hanson, 1984; Reddy et al., 1988; Sagiv et al., 1985). However, there was considerable individual variations in the hemodynamic responses among these patients, which could not be predicted by resting hemodynamics, LVEF or functional classification (Elkayam et al., 1985; MeKelvie et al. 1995).

Safety of Resistance Training in Cardiac Rehabilitation Programs

Haslam and associates (1988) assessed electrocardiographic (ECG) and direct arterial BP responses during single-arm, single-, and double-leg lifting at 20, 40, 60 and 80% of 1-RM in low risk cardiac patients with normal LV function (EF ≥ 50%). None of the weightlifting exercises resulted in clinically significant ST-segment depression, angina or ventricular arrhythmias. Only single-leg lifting at 80% of 1-RM, and double-leg lifting at 60% and 80% of 1-RM produced RPP values that exceeded those attained during cycle ergometer testing at 85% of maximal aerobic capacity. The values demonstrated higher HR and BP responses during lower body resistance exercise compared to upper body exercises, due to the larger muscle mass (Gobel et al., 1977; MacDougall et al., 1985; MacDougall et al., 1992; Misner et al., 1990; Verrill and Ribisl, 1996). It should be noted that although the RPP at high weightlifting intensities can rise to higher levels than during aerobic exercise at 85% of VO_2max, the increased myocardial oxygen demand is usually maintained for less than 30 seconds during weightlifting compared with aerobic exercise where the demand lasts for several minutes. Therefore, light to moderate weightlifting exercise can be considered safe for low risk cardiac patients, who have a low risk for the development of LV dysfunction (Haslam et al., 1988).

Circuit weight training (CWT) is an exercise method for strength development. It incorporates a series of selected weight training exercises that are performed in a sequence or in a "loop". With circuit training, one performs approximately 12-15 repetitions using about 40-60% of 1-RM, on specialized weight machines. The individual moves from one weight machine to another with short rest periods between stations (15-30 seconds) (Butler et al., 1987; Gettman et al., 1978; Gettman and Pollock, 1981; Pollock and Wilmore, 1990). The fact that CWT can improve strength, body composition, and also to a lesser extent cardiorespiratory endurance, makes this form of exercise an appealing addition to a cardiac rehabilitation program (Butler et al., 1987; Gettman and Pollock, 1981).

A large amount of evidence on the safety and efficacy of CWT, in stable cardiac patients previously participating in cardiac rehabilitation programs, has been gathered in the past two decades (Butler et al., 1987; Faigenbaum et al., 1990; Saldivar et al., 1983; Sparling and Cantwell, 1989; Stralow et al., 1993; Vander et al., 1986). Butler and associates (1987) compared LV wall motion responses in CWT (two circuits at 40-60% of 1-RM) with aerobic exercise (35 minutes of treadmill exercise at 85% of maximal HR). A decline in segmental wall motion was demonstrated in five of 61 LV wall segments during aerobic exercise, but only in one segment during CWT. Faigenbaum and coworkers (1990) demonstrated that HR values attained during 1-RM testing and circuit weight trials at 75% of MVC, were 54% and 58% lower, respectively, than the HR values attained during the maximal graded exercise test (GXT). Moreover, the mean peak RPP values recorded during the GXT were significantly higher compared to the RPP achieved during the 1-RM testing and CWT. The data of Stralow et al. (1993) are in agreement with previous studies which found lower mean peak HR, SBP and RPP during strength training compared to their respective responses at 85% of maximal aerobic capacity during a treadmill GXT. Crozier-Ghilarducci and colleagues (1989) studied the effect of high resistance training intensity in stable cardiac patients. The investigators showed that weight training at 80% of MVC was safe and efficacious in this low risk patient population. Even though the program consisted of high intensity resistance training, the HR attained ranged from 45 to 64% of the maximal treadmill HR. Furthermore, subjects did not show ST-segment abnormalities nor angina pectoris during exercise (Crozier-Ghilarducci et al., 1989). Thus, the above studies provide evidence that CWT can be safely performed by stable low risk cardiac patients (Butler et al., 1987; Crozier-Ghilarducci et al., 1989; Faigenbaum et al., 1990; Haslam et al., 1988; Sparling et al., 1990; Stewart et al., 1995).

Blood pressure measurements in cardiac patients which were collected before, during, and after CWT, demonstrated a slight increase or no change compared to resting values (Crozier-Ghilarducci et al., 1989; Saldivar et al., 1983; Sparling et al., 1990). Circuit weight training BP values were lower (Butler et al., 1987; Faigenbaum et al., 1990; Saldivar et al., 1983) or slightly higher (Crozier-Ghilarducci et al., 1989; Sparling et al., 1990; Squires et al., 1991; Stralow et al., 1993) compared to dynamic exercise at 85% of maximal aerobic capacity BP measurements. However, these data should be interpreted with caution since BP, which was measured (auscultation method using bladder cuff) immediately after each set of exercise, could not have reflected the BP attained during the actual exercise set, when the BP value is much higher (Haslam et al., 1988; MacDougall et al., 1985; Wiecek et al., 1990). Wiecek and associates (1990) compared direct and indirect measures of systemic arterial BP during weightlifting in CAD patients. Indirect SBP both at rest and during leg press resistance exercise were 13% less than the SBP recorded directly. Mean indirect SBP recorded immediately after exercise was 31% lower than values recorded directly during the actual lift. Diastolic BP at rest and during lifting was similar using either method. The highest direct pressure value was measured during the final repetition. Both DBP and SBP rapidly decreased to resting values (within 5-15 seconds) after completing the lift (Wiecek et al., 1990). Therefore, because of the rapid drop in BP that occurs after weightlifting exercise, indirect measurements immediately after resistance exercise do not represent accurate information regarding the arterial BP generated during lifting.

Ehsani et al. (1982) demonstrated that cardiac patients who participate for a long time in a Phase IV community based cardiac rehabilitation program can engage safely in moderate to heavy resistance training in addition to their aerobic activity. The authors studied the effects of intense and prolonged aerobic exercise training on LV function in patients with CAD. Training consisted of endurance exercise three times per week at 50-60% of VO_2max for 3 month, followed by aerobic exercise 4-5 days per week at 70-80% of VO_2max for 9 months. Echocardiographic examination during isometric exercise at 40% and 60% of MVC were performed before and after the training period. Before training, LV fractional shortening and mean velocity of circumference of shortening decreased progressively in response to isometric handgrip exercise, suggesting a decline in

LV function. Such reduction in LV fractional shortening and mean velocity of circumference shortening was not observed in the post training period. At comparable levels of mean BP, mean velocity of circumference shortening was significantly higher after training, suggesting improvement in LV function during isometric exercise (Ehsani et al., 1982). One needs to take into account that these data were obtained from a small selected group of highly motivated patients; an equivalent response may not be demonstrated by the general population of cardiac patients. However, the results indicate that prolonged and vigorous endurance exercise results in peripheral and central adaptations which are characterized by an improvement in LV performance.

With the increased documentation of the safety and efficacy of CWT found with cardiac patients participating in phase III outpatients cardiac rehabilitation programs, attempts were made to determine the hemodynamic responses and feasibility of low level weight training during early outpatient (phase II) cardiac rehabilitation programs (Daub et al., 1996; Squires et al., 1991; Stewart et al., 1995). These studies included patients with reduced LV function, i.e. EF ≥ 35%. Stewart et al. (1995) studied 2-D Echo/Doppler and clinical responses in men starting cardiac rehabilitation as soon as 2 weeks after acute myocardial infarction. After 2 weeks of usual care, patients were randomly assigned to either a control or to a CWT group. The control group continued usual care that consisted of cycling exercise 20-25 minutes 3 days per week. The CWT group exercised at 40% of MVC in addition to 10 minutes of cycling for 10 weeks and performed 2 sets of 6 exercises. Mean wall motion scores for 130 segments did not differ between the two groups at baseline or after training. In another study no evidence of clinical complications or ECG signs of ischemia were found in patients participating in weight a training program 38 ± 13 days after a cardiac event (Squires et al., 1991). Thus, no adverse cardiovascular responses have occurred in properly selected patients participating in weight training in addition to aerobic exercise training soon after myocardial infarction (Daub et al., 1996; Squires et al., 1991; Stewart et al., 1995). However, data is lacking regarding the hemodynamic responses during resistance exercise in many subgroups of LVD patients. Furthermore, there is no sufficient information comparing cardiovascular response during upper body vs. lower body resistance exercises in LVD patients.

Health Benefits of Resistance Training for Cardiac Patients

Resistance exercise contributes to better health by preventing musculoskeletal disorders, increasing muscle strength and bone mineral density, helping to maintain desirable body composition, and improving self image and self efficacy (Stewart, 1989; Stone et al., 1991). For the last two decades evidence has emerged illustrating that resistance training has a beneficial effect on some CAD risk factors comparable to the effect of endurance training (Hurley and Kokkinos, 1987; Hurley et al., 1988).

Several studies demonstrated that strength training, in the form of CWT, seems to produce similar beneficial effects on BP as endurance training (Harris and Holly, 1987; Kelemen et al., 1986; Kelemen et al., 1989; Stewart et al., 1989). Notwithstanding, the results are controversial. Smutok et al. (1993) compared the effect of strength against aerobic training program in middle-aged men with risks for CAD. No change was found in resting BP in both groups. Nevertheless, none of the CWT studies resulted in a exacerbation of resting BP (Cononie et al., 1991; Harris and Holly, 1987; Kelemen et al., 1986; Kelemen et al., 1989; Smutok et al., 1993; Stewart et al., 1989). Therefore, it can be concluded that CWT can be performed safely by patients with mild hypertension.

It appears that resistance training may improve some risk factors for CAD such as: increasing HDL-cholesterol, lowering LDL-cholesterol, improving glucose regulation, and increasing insulin sensitivity. Despite the reports of an improved lipid profile from resistance training, many design limitations prevent consistent conclusions regarding the use of resistance training as a tool for this specific risk factor intervention. Some of the studies above lacked a control group (Goldberg et al., 1984; Ulrich et al., 1987), pre- and post-training blood samples (Goldberg et al., 1984; Ulrich et al., 1987), diet monitoring (Ulrich et al., 1987), measurement of change in body composition (Goldberg et al., 1984; Ulrich et al., 1987), or used subjects with a low lipid risk profile for the development of CAD (Goldberg et al., 1984; Hurley, 1989; Ulrich et al., 1987).

Studies in young and middle-aged healthy subjects (Fluckey et al., 1994; Hurley et al., 1988; Miller et al., 1994), subjects considered at high risk for CAD (Smutok et al., 1993), and non-insulin-dependent diabetes mellitus patients (Fluckey et al., 1994) have demonstrated an improvement in glucose tolerance and insulin sensitivity after engaging in resistance training exercise. However, more controlled resistance training studies are needed in order to establish these potential benefits especially in the cardiac population. Information regarding training thresholds necessary to evoke changes in risk factors, the optimal degree of resistance weight, number of repetitions, number of sets of exercise and the length of the rest interval between sets are not well known. Although aerobic training has been clearly proven to improve CAD risk factors in cardiac patients, the magnitude of the direct effect of resistance training on CAD risks factor is less well defined.

Progressive resistance exercise increases strength and muscle mass, consequently, individuals who participate in long-term weightlifting exercise display muscle hypertrophy (Gettman et al., 1978; Partely et al., 1994; Tesch, 1988; Wilmore et al., 1976). Studies in cardiac patients have demonstrated increased muscle mass with no significant change in body weight or percentage of body fat (Crozier-Ghilarducci et al., 1989; Sparling et al., 1990; Stewart et al., 1988). Crozier-Ghilarducci et al. (1989) showed an 11% increase in quadriceps girth in cardiac patients following 10 weeks of resistance training at 80% of 1-RM, however, body weight and body fat remained unchanged.

Pronounced loss of the mineral and collagen matrices of bone occur around the fifth decade in both genders, resulting in enhanced bone susceptibility to fractures (Marcus, 1991; Menkes et al., 1993). Cross-sectional studies have demonstrated an increase in bone mineral density (BMD) and in bone mass in physically active subjects compared to sedentary age matched persons (Block et al., 1989; Bouxsein and Marcus, 1994; Dalen and Olson, 1974; Helela, 1969). However, it seems that different modes of exercise produce different adaptation responses. Hamdy et al. (1994) demonstrated greater gain in upper limb bone mass in adults engaging in weight-lifting exercise compared to individuals performing endurance activities such as running and recreational exercises. Furthermore, studies have shown that resistance exercise training appears to attenuate the normal bone loss associated with aging and can even lead to small increases in BMD and bone mass (Hughes et al., 1995; Hurely, 1994; Menkes et al., 1993; Wilmore, 1991). The increase in BMD is associated with increased muscular strength (Hughes et al., 1995), which in return, can improve the capacity to perform activities of daily living (Frontera et al., 1990).

Effects of Resistance Training on Muscular Strength

It is well known that participating in a weight training program results in an increase in muscular strength and endurance (Gettman et al., 1978; Kass and Castriotta, 1994). A stronger musculature may reduce the relative stress imposed by occupational and recreational activities of daily living (Stone et al., 1991). Furthermore, increased muscular strength results in increased absolute muscle force output, and increased tissue strength such as tendons and ligaments (Stone, 1988; Stone et al., 1991). This strengthening effect can lessen the likelihood of musculoskeletal injuries which often accompany physical activity. Therefore, improved muscle strength results in increased functional capacity, which can lead to more independent living and to enhanced quality of life.

Healthy individuals and athletes have been shown to improve their muscular strength and endurance after engaging in CWT programs (Gettman et al., 1978; Wilmore et al., 1978). Studies have demonstrated increases in strength ranging from 20-45%. Comparable results have been seen in stable cardiac patients who participated in CWT during cardiac rehabilitation programs (Crozier-Ghilarducci et al., 1989; Kelemen et al., 1986; McCartney et al., 1991; Sparling et al., 1990; Stewart et al., 1988). Kelemen et al. (1986) documented a 24% increase in muscular strength in the CWT group while no change was observed in muscle strength in the control patients. Sparling et al. (1990) found a 22% increase in strength for all 12 exercises in cardiac patients after 6 months of weight training at 30% to 40% of 1-RM.

Along with the increase in muscular strength and endurance following resistance training, any absolute submaximal work load would require a lower comparable effort and consequently be perceived as less strenuous (McCartney et al., 1991). Since most activities of daily living require less than a maximal effort, patients who weight train will be able to perform strenuous daily activities at a diminished percent of maximum and perception of effort. This improvement will result in enhanced quality of life and decreased risk for musculoskeletal injuries (McCartney et al., 1991; Stewart, 1989).

Effects of Resistance Training on Aerobic Performance

Resistance training can produce a small increase in aerobic capacity in healthy adults of all ages including the elderly. This increase is primarily associated with an increase in muscle mass, but not necessarily improved cardiorespiratory central function (Fiatarone et al., 1990; Frontera et al., 1990; Hickson et al., 1980; Kass and Castriotta, 1994). A study in frail deconditioned elderly demonstrated lower extremity muscle adaptations (strength and size) to high intensity strength training. The increase in strength ranged from 61 to 374% over baseline, which was coupled with a 48% improvement in tandem gait speed (Fiatarone et al., 1990). Moreover, Frontera et al. (1990) showed an increase in maximal aerobic capacity during leg cycle ergometry testing in elderly subjects involved in a high intensity resistance training program for the lower body. The increased aerobic capacity was due to local adaptation in the trained muscle (i.e. increased muscle strength and mass, increased oxidative enzyme concentrations and greater capillary density), since such a phenomenon was not observed during arm cycle ergometry.

Aerobic performance of patients with cardiac disease and elderly persons can be limited by leg fatigue resulting in termination of exercise without coexisting evidence of cardiorespiratory limitations (Hung et al., 1984; McCartney et al., 1989; McCartney et al., 1991; Oldridge et al., 1989). Therefore, patients with weak leg muscles will gain additional benefits if resistance training is coupled with their conventional endurance training program. Increased leg muscle strength will allow patients to engage in aerobic modalities for longer periods of time. McCartney et al. (1991) reported an average increase of 29% in 1-RM strength in stable cardiac patients who engaged in combined aerobic and resistance training, compared to an average increase of 8% in the group that performed aerobic training alone. The cycling time at 80% of initial maximal power output before attaining a Borg rating of very severe, increased by only 11% in the aerobic training group compared to 109% in the combined training group. In addition, maximal exercise capacity on the cycle ergometer increased by 15% in the combined group compared with a 2% increase in the aerobic control group. Therefore, in stable low risk cardiac patients, combined aerobic and resistance exercise is a more effective method of increasing aerobic performance and strength than traditional endurance training alone (Kelemen et al., 1989; McCartney et al., 1991).

Increased muscular strength and endurance result in improved performance of endurance activities (such as walking or climbing up stairs), thus, facilitating some everyday physical activities. In cardiac patients who are severely deconditioned, resistance training can cause muscular changes that may lead to enhanced ability to engage in aerobic exercise, which consequently will result in improved aerobic performance.

In summary, resistance training can produce several beneficial adaptations that result in favorable changes in CAD patient's risk profile. Some of the data are more conclusive such as the beneficial effect of resistance training on glucose metabolism. However, more controlled studies on the effect of resistance training on blood lipid profile or resting BP are warranted, mainly in the cardiac population. Nevertheless, the beneficial effect of resistance training on promoting independent lifestyle and enhanced quality of life via increased muscle mass and strength is well documented, emphasizing the importance of this type of exercise in cardiac rehabilitation programs.

Patient Screening and Consideration

During the late 80s and early 90s, the conventional inclusion criteria of cardiac patients for a resistance training program were mainly directed toward low risk patients already participating in a traditional aerobic exercise program for at least 3 months (Franklin et al., 1991; Kelemen, 1989; McKelvie and McCartney, 1990; Sparling and Cantwell, 1989). In addition, patients were at least 4 months post myocardial infarction or coronary artery surgery before they were allowed to participate in a resistance training program (Kelemen, 1989; Sparling and Cantwell, 1989).

Generally, exclusion criteria for resistance training resembled those used for any outpatient cardiac rehabilitation program, i.e. phase III-IV. In many cardiac rehabilitation programs the exclusion for CWT were similar to those of aerobically oriented activities (Franklin et al., 1991; Sparling and Cantwell, 1989). Patients were excluded for the following reasons: unstable angina, uncontrolled hypertension (systolic BP > 160 mmHg or diastolic BP >100 mmHg), uncontrolled arrhythmias, a recent history of congestive heart failure, a maximal aerobic capacity of less than 6-7 metabolic equivalents (METs) (1 MET = $3.5\ \mathrm{ml \cdot kg^{-1} \cdot min^{-1}}$) during symptom limited GXT, or LV dysfunction (EF < 45%) (Franklin et al., 1991; Kelemen, 1989; Sparling and Cantwell, 1989; Verrill et al., 1992). Both the cardiac rehabilitation program director and the patient's personal physician should assess and approve the patient's participation in the resistive training program (Franklin et al., 1991; Verrill et al., 1992); persons qualifying for resistance training should begin exercise in a supervised setting.

However, recent studies performed in early outpatient cardiac rehabilitation settings (phase II) as soon as 2 weeks after acute myocardial infarction demonstrated no adverse cardiovascular responses in properly selected patients participating in CWT at 40% of MVC (Daub et al., 1996; Squires et al., 1991; Stewart et al., 1995). In light of these findings the revised Exercise Standards of the American Heart Association (AHA) (AHA, 1995), American Association of Cardiovascular and Pulmonary Rehabilitation (AACVPR) (AACVPR, 1995) and recent American College of Sports Medicine (ACSM) (ACSM, 1995) guidelines for exercise testing and prescription include much less conservative indications for resistance exercise training for cardiac outpatients. Initial resistance training activities can be introduced to patients during the first 2 weeks of an outpatient program. Later in the program if the patients are medically stable they can be allowed to participate in a regular resistance training program. ACSM published specific indications for resistance training for cardiac outpatients for the first time in 1995 (ACSM, 1995). The inclusion criteria consists of:

(a). minimum of 4 to 6 weeks after myocardial infarction (MI) or coronary artery surgery,
(b). minimum of 4 to 6 weeks in supervised aerobic program or completion of Phase II,
(c.) minimum 1 to 2 weeks following PTCA or other revascularization procedures without MI,
(d). diastolic pressure < 105 mmHg,
(e). peak exercise > 5 METs, and
(f). not compromised by CHF, unstable symptoms, or arrhythmia.

Low to moderate risk patients who in the past were excluded from a resistance training regimen are capable of exercising safely with resistance using lighter load. Such patients include older cardiac patients, patients with reduced left ventricular function (EF > 35%), patients with mitral valve prolapse syndrome and heart transplant patients (Braith et al., 1993; Braith et al., 1994; Braith et al., 1996; Daub et al., 1996; Frederickson, 1988; McKelvie et al., 1995; Munnings, 1993; Verrill and Ribisl, 1996).

Exercise Guidelines

In recent years, the AHA (AHA, 1995) ACSM (ACSM, 1990; ACSM, 1995) and AACVPR (AACVPR, 1995) have emphasized the importance of a comprehensive training program. They espouse an overall exercise program for developing and maintaining cardiorespiratory fitness, body composition, and muscular strength and endurance in both the healthy adult and in the majority of subjects with heart disease.

Before entering a resistance training program, each patient should be briefed on the proper technique and safety rules of resistive training. Instruction and demonstration should include correct body position, speed of movement, range of motion and proper breathing pattern (Franklin et al., 1991; Sparling and Cantwell, 1989). Initial resistance training activities can be introduced to the patients during the first 2 weeks of an outpatient program, which may include the use of 1-3 kg dumbbell weights, light hand weights, and/or resistive tubing. Six weeks into the program, functional capacity assessment (i.e. symptom limited GXT) and risk stratification of the patients are made. Thereafter into the program, the patients can be allowed to engage in a regular weight training program, e.g., weight machines used as a single station or CWT.

In order to establish the initial weight load, 1-RM testing is recommended. This type of strength testing is most efficacious for evaluating maximal strength (AACVPR, 1995; ACSM, 1995; Franklin et al., 1991). Injuries related to 1-RM strength testing are rare and derive primarily from previous orthopedic problems (Pollock et al., 1991). Shaw et al. (1995) evaluated injuries associated with 1-RM testing in the elderly. Out of 83 subjects (65.8 ± 6.2 years) only 2 subjects sustained an injury (2.4% of total subjects), whereas 81 subjects (97.6% of total) completed the 1-RM assessment without harm. One-repetition maximum tests have been found hemodynamically safe in healthy adults. Of 6,653 subjects none experienced a clinically significant, nonfatal or fatal cardiovascular event in association with 1-RM strength testing (Gordon, 1995). In addition, 1-RM strength testing was found to be safe in low risk cardiac patients as HR and mean peak RPP values attained during 1-RM testing were significantly lower compared to those achieved during GXT (39, 42). However, a more conservative approach for initial weight establishment can be applied by determining the maximal load that the patients can lift twice. This method of testing is assumed to be 90% of 1-RM. Using this 90% value, a 1-RM is calculated and used to establish the training weights (AACVPR, 1995; Franklin et al., 1991; Kelemen, 1989; Sparling et al., 1990).

ACSM, AACVPR and AHA recommendations for resistance training consist of 8-10 exercises which train the major muscle groups of the body, one set of 10-15 repetitions at a load of 30%-50% of the 1-RM for each exercise, performed 2-3 days per week (AACVPR, 1995; ACSM, 1995; AHA, 1995). Once 15 repetitions can be comfortably completed by the patient the load can be raised by an additional 5% (ACSM, 1995; AHA, 1995; Sparling and Cantwell, 1989). Cardiac rehabilitation settings that do not use 1-RM as a prescribing reference, should initially choose a weight load that will allow cardiac patients to exercise at RPE level of 12-13, and later on to increase weight load until reaching RPE sensation of 15. These guidelines are based on the literature that supports prescribing single set of exercise to fatigue for developing muscular strength (Feigenbaum and Pollock, 1997; Messier and Dill, 1985; Starkey et al., 1996; Stowers et al., 1983). Starkey and associates (1996) demonstrated that 1 set performed to volitional fatigue (8-12 repetitions) was as effective as 3 sets for increasing knee extension and knee flexion strength and muscle thickness in previously untrained adults. In another study Braith et al. (1989) evaluated the effectiveness of resistance training performed either 2 days per week or 3 days per week. The authors found that adult exercisers who perform a single set of bilateral knee extension to volitional fatigue 2 days per week can derive approximately 80% of the benefits achieved by training 3 days per week. Based on these data, a well rounded training program can be attained encompassing cardiorespiratory fitness, and muscular strength and endurance, that is cost efficient and not highly time-consuming.

An important element of exercise safety in cardiac rehabilitation programs is the stratification of patients according to their risk for acute cardiovascular complications during exercise and overall prognosis. Risk status is related to the type and pathophysiologic severity of the cardiovascular disease, the degree of LV dysfunction and exercise-induced myocardial ischemia as manifested by ST segment depression and/or angina pectoris (AACVPR, 1995; ACSM 1995). Stratifications criteria for cardiac patients based on AACVPR (1995) and ACSM (1995) are as follows:

Low risk patients. No significant LV dysfunction (EF≥ 50%). No resting or exercise-induced ischemia or arrhythmias. Uncomplicated myocardial infarction, coronary artery bypass graft (CABG), angioplasty, arthrectomy. Functional capacity above 6 METs 3 weeks after cardiac event.

Moderate risk patients. Mild to moderate depressed LV function (31 ≤EF≤ 49%). Exercise-induced myocardial ischemia. Functional capacity ≤ 5-6 METs 3 weeks or more after cardiac event.

High risk patients. Severely depressed LV function (EF≤ 30%). Complex ventricular arrhythmias at rest, or appearing or increasing with exercise. Marked exercise-induced myocardial ischemia. Exertional hypotension (≥ 15 mmHg decrease in SBP during exercise. Myocardial infarction complicated by CHF, cardiogenic shock, and/or complex ventricular arhythmias. Survivor of cardiac arrest.

Summary

Reduced myocardial function results in a complex manifestation of chronic responses involving autonomic nervous system, endocrine organs, skeletal muscle, kidneys and regional vascular beds; resulting in clinical symptoms of fatigue and dyspnea. The latter is followed by physical inactivity leading to skeletal muscle atrophy and weakness. Endurance training in LVD patients, results in increased aerobic capacity through peripheral adaptation. The improvement in functional capacity in these patients can result in a major impact on their quality of life. Since LVD patient's symptoms, such as tiredness, dyspnea with exertion, and overall weakness are most common, engaging in resistance training may result in muscular changes that can lead to improvement in their aerobic performance via increasing muscle mass and strength.

Resistance exercise has been previously regarded as hemodynamically hazardous for patients with cardiovascular disease or for those with high risk factors for a future cardiac event. Consequently, resistance exercise has been traditionally discouraged in cardiac rehabilitation programs due to the assumption that the increased BP response seen in this form of exercise, imposes an additional risk to cardiac patients. However, many daily and vocational activities require that patients with cardiac disease perform tasks that involve lifting and straining. Therefore, it is important to recognize that the cardiac patient requires a minimum threshold level of strength for daily living activities, equivalent to those of a healthy individual.

Recent evidence suggests that resistance exercise may be less hazardous than was once presumed, especially in low risk cardiac patients. Investigators have been able to demonstrate that light to moderate weightlifting exercise can be considered safe for these cardiac patients and that the risk of developing compromised LV function is less during weightlifting compared to conventional clinical aerobic exercise tests. Furthermore, the increased DBP seen during resistance exercise provides a protective effect by increasing coronary perfusion pressure. This increase in coronary perfusion pressure improves subendocardial BF, resulting in a reduction of the development of myocardial ischemia. Resistance exercise contributes to better health by preventing musculoskeletal disorders, helping to maintain desirable body composition, and improving self image and self efficacy. With the increase in muscle strength after training, daily tasks will be perceived as less strenuous, resulting in a more independent lifestyle and enhanced quality of life. The increased leg muscle mass and strength due to resistance training may improve aerobic capacity. In cardiac patients who are severely deconditioned, resistance training can cause muscular changes that can lead to the enhanced ability of these fragile patients to engage in aerobic exercise, thus improving their aerobic capacity.

In recent years, current trends have emphasized the importance of a comprehensive exercise training program for developing and maintaining cardiorespiratory fitness, body composition, and muscular strength and endurance in the healthy adult and the majority of subjects with heart disease. AHA, AACVPR and ACSM have developed guidelines for resistance training in low risk cardiac population. The recommendations include the following: 1-RM as the testing procedure, training consisting of 8-10 exercises which train the major muscle groups of the body, 10-15 repetitions at a load of 30%-50% of the 1-RM for each exercise, and a frequency of 2-3 days per week. Once 15 repetitions can be accomplished, the weight can be increased by an additional 5%.

The AACVPR, AHA and ACSM guidelines for resistance exercise in low risk cardiac patients are based on the guidelines previously developed for healthy adults. However, the training intensity for cardiac patients is lower (moderate fatigue vs. maximal effort), and the number of repetitions are higher (10-15 vs. 8-12) than is recommended for healthy adults. There are no specific guidelines for strength testing or resistance exercise training for low-moderate risk cardiac patients with LVD due to insufficient data on safety. Therefore, the importance of the present study is to add more information that could help in prescribing a safe resistance training exercise program to low-moderate risk cardiac patients.

CHAPTER 3
METHODS

Subject's Characteristics

Fifteen patients (n=15 males) (from two cardiac rehabilitation programs) volunteered to participate in the study: the Zinman College Cardiac Rehabilitation Program at Wingate Institute and from the Cardiac Rehabilitation Center in Tel Aviv, Israel. All patients had documented coronary artery disease (CAD) determined by at least one of the following: 1) history of prior myocardial infarction (MI); 2) history of coronary artery bypass graft surgery (CABG); and 3) demonstration on angiography of CAD determined by a minimum stenosis of 70% in at least one vessel. Each subject had been previously diagnosed with left ventricular dysfunction (LVD) with an ejection fraction (EF) range between 30 to 49% by means of echocardiography or angiography procedures. The patient's age averaged 65±6.5 years (mean±SD) (range 50-74 years). All subjects had been participating in a cardiac rehabilitation program, training aerobically a minimum of twice a week between 3 months up to 6 years (2.5 ± 0.8 yrs).

Prior to taking part in the study, patients underwent preliminary screening by the cardiologist of the Cardiac Rehabilitation Program at the Zinman College. Inclusion criteria for participating in the study included: 1) age 50 to 75 years; 2) stable medical condition; 3) New York Heart Association (NYHA) classification I and II: CAD without symptoms at rest and with/without symptoms during ordinary activity; 4) functional capacity > 5 METs; and 5) optional drug therapy (digoxin, diuretics, ACE-inhibitors, beta-blockers, anti-anginal agents, etc.). Contraindication to participation in the study included: 1) acute unstable myocardial ischemia; 2) angina at rest/ or exercise < 5 METs; 3) cardiac event within past 3 months; 4) other diseases that would interfere with the completion of the study (i.e. thyrotoxicosis, uncontrolled hypertension or diabetes mellitus, anemia, lung diseases or renal failure, primary valvular heart disease; 5) patients with a recent (within 6 month) cerebral vascular event; 6) patients with orthopedic problems and/or peripheral vascular disease that would limit exercise; and 7) resting blood pressure (BP) 160/100 mmHg or above. All patients continued taking their usual daily prescribed medications during the study and on days of the experiment. Medications included: nifedipine (4 pts), dilitiazem (2 pts), verapamil (2 pts), atenolol (3 pts), propranolol (1 pt), metoprolol (1 pt), captopril (5 pts), enalopril (2 pts), isosorbide (6 pts), furosemide (4 pts), hydrochlorothiazide (2 pts), digoxin (2 pts) and amiodarone (1 pt).

All experiments took place in the Cardiac Rehabilitation Program Laboratories at the Zinman College, Wingate Institute, Israel. In order to avoid diurnal effect, all subjects completed experimental procedures at the same time of the day. All experiments were performed in an air conditioned room at 24C°-25C°, 60% to 63% relative humidity and 758-763 mmHg barometric pressure. All subjects were asked to refrain from any exercise training for at least 24 hours before experiments, abstain from drinking alcohol 48 hours prior to testing, and report to the laboratory at least 4 hours post caffeine consumption and 2 hours postprandial. To help verify these standardized conditions subjects were asked to complete a 24 hour health history and activity questionnaire prior to each visit (Appendix A).

Study Design

The study consisted of five visits lasting 1-1.5 hours in which patients perform two separate experiments. Experiment 1 assessed cardiovascular hemodynamic and left ventricular physiologic responses to strength testing in LVD patients. Experiment 2 assessed cardiovascular hemodynamic and left ventricular physiologic responses to a single bout of resistance exercise performed at varying intensities of effort. The sequence of tests on the first visit (visit 1) included: medical evaluation, body composition, resting and diagnostic echocardiography combined with a treadmill symptom limited graded exercise test (SL-GXT). During visit 2 peak oxygen consumption (VO_{2peak}) was determined during treadmill SL-GXT. During the third visit (visit 3) subjects performed one-repetition maximum (1-RM) of one-arm biceps curl (BIC) and knee extension (KE) tests. For the 4th and 5th visits, subjects were assigned to perform exercise bouts of 10-15 repetitions at 20%, 40%, and 60% submaximal work loads based on their previously determined 1-RM testing protocol. Patients rested 5 days between visits.

Visit 1: Experimental Protocol

All patients received a comprehensive explanation of the proposed study, its benefits, inherent risks and expected commitment with regard to time. Following explanation of the proposed study, all patients were allowed a period of questioning. For the protection of human subjects, the protocol of the study was approved by the Institutional Review Board at the University of Florida, USA and by the Human Research Committee of the Zinman College at Wingate Institute, Israel. Those patients who agreed to participate were required to sign an informed consent (Appendix B). The subjects then completed a medical evaluation by the cardiologist of the Cardiac Rehabilitation Program at the Zinman College, and those patients who were included in the study then continued with their first visit testing.

Body Composition

Body composition was assessed from the sum of four skinfold sites: biceps, triceps, subscapular and iliac crest (Durnin and Womersley, 1974), utilizing a Skyndex®/System I Electronic Body Fat Calculator caliper (Caldwell, Justiss & Co. Inc., Fayetteville, AR). Subcutaneous fat was measured by grasping a skinfold of fat with moderate pressure by the thumb and the forefinger. The caliper was placed approximately 1 cm perpendicular to the fold, then the caliper tips were released over the skinfold. The value of the skinfold thickness was entered into the calculator memory where it was automatically recorded and calculated. Body density (D_b) was predicted for males over the age of 50 using the Durnin-Womersley equation (Durnin and Womersley, 1974):

$$D_b = 1.1715 - 0.0778 \log(\Sigma \text{ skinfolds}).$$

Following the calculation of D_b the Siri equation (Siri, 1961) was used to estimate body fat percentage:

$$\% \text{ fat} = 495/ D_b - 450$$

Weight to the nearest 0.1 kg was measured on a digital weight scale Shekel TCS 155 (Shekel, Beit Keshet, Israel). Height to the nearest 0.1 cm was measured with a wall mounted meter scale Shekel TCS 155 (Shekel, Beit Keshet, Israel).

Echocardiographic Measurements

Resting and diagnostic echocardiography were performed in order to verify the LVEF and screen for potential exclusionary factors. For each echocardiography evaluation, complete two-dimensional (2-D) echocardiography was performed using standardized methodology and commercially available equipment (Vingmed 800 A Sonotron and Interspec AT Apogee transducer 2.25 and 3.25 MHz, Horten, Norway). Two-dimensional and motion (M)- mode echocardiographic measurements were performed at rest with subjects in the left lateral decubitus position in order to obtain images in multiple cross-sectional planes for assessment of chamber sizes and left ventricular systolic function, using the following views 1) parasternal long-axis; 2) parasternal short axis; 3) apical 4- and 5-chamber view and 4) apical 2-chamber view. Complete pulsed and high repetition frequency and/or continuous wave, when required, Doppler examination was also performed to determine the presence and severity of valvular disease. Following a review of the images for exclusion criteria (such as hemodynamically significant primary valvular heart disease), each subject completed a treadmill SL-GXT (as detailed below). Following the SL-GXT, the patient immediately resumed the left lateral decubitus position and echocardiographic images were obtained within 30 seconds of cessation of exercise (Robertson et al, 1983).

Left ventricular end diastolic dimensions (LVEDD) were determined as the distance from the leading edge of the left side of the interventricular septum to the leading edge of the posterior endocardium of the left ventricle, at the peak of the R wave on the simultaneously recorded electrocardiogram (ECG). Left ventricular end systolic dimensions (LVESD) were taken as the vertical distance from the maximal excursion of the left ventricular endocardium echocardiography during systole to the interventricular septum. Left ventricular end diastolic volume (LVEDV) and LV end systolic volume (LVESV) were obtained from the 2-D echocardiographic images in the apical, four chamber view using the modified Simpson's rule algorithm (Albin and Ranko, 1990).

Calculations. Stroke volume (SV), cardiac output (CO) and EF were calculated with the following equations: SV = left diastolic volume - left systolic volume, CO = heart rate (HR) x SV and EF = SV divided by LVED. Rate pressure product was derived from the product of HR times the SBP. Mean arterial blood pressure (MAP) was calculated as DBP plus one-third of pulse pressure. Total peripheral resistance (TPR) was calculated by dividing MAP by CO. Peak SBP/LVESV ratio was determined by SBP divided by LVESV.

For analyzing regional LV function the LV wall was divided into seven segments as follows: apex, basal, septal, anterior, posterior, lateral and inferior. Each segment was given a numeric value that indicated a wall motion pattern. The wall motion scores included: 0 = normal, 1 = hypokinesis, 2 = akinesis and 3 = dyskinesis. The apical 4-chamber view was divided into 4 segments; apex, septum, basal and lateral wall. Long-axis view was divided into septum, basal and posterior wall. Short-axis was divided into septum, lateral and inferior wall and 2-chamber view was used to assess the anterior wall region (Appendix C). The echocardiographic views for each patient were placed onto a quad-screen format for simultaneous viewing, after which they were transferred onto a videotape in a continuous sequence for viewing during the rating process. The wall motion scores were assigned to each LV segment by a cardiologist.

Diagnostic Graded Exercise Test

Upon finishing the resting echocardiographic measurements the subjects were then prepared to perform a SL-GXT on a Quinton Club Track 3.0 treadmill (Quinton®, Seattle, WA). All SL-GXTs were supervised by the cardiologist of the Zinman College Cardiac Rehabilitation Program. A crash cart with all essential emergency medications, supplemental oxygen and a defibrillator were stationed near the treadmill during every test.

The modified Naughton protocol was used for the treadmill SL-GXT. The protocol involves a constant speed of 2 mph, beginning at 0% grade, increasing 1 MET (3.5% grade) every 2 minutes (Pollock and Wilmore, 1990). A 12-lead ECG and HR were recorded at one minute intervals using Cardiofax model 3353/D/F/L (Nihon Kohden, Tokyo, Japan), and was monitored continuously at rest, exercise and recovery periods of each test. Blood pressure (BP) was measured by auscultation using Aneroid Sphygmomanometer (Nihon Kohden, Tokyo, Japan) during rest, 30 seconds prior to the end of each 2 minute stage of exercise, at peak exercise, 1, 3, 5 and 7 minutes of recovery. The HR and BP values which were measured standing prior to mounting the treadmill were considered as baseline value criteria.

Subjects were verbally encouraged to continue exercise as long as they could. Rating of perceived exertion (RPE) using the Borg scale (Borg, 1978) was recorded during each minute of exercise. The SL-GXT was terminated upon subject's request, or if one of the following clinical indications appeared prior to volitional fatigue: 1) progressive angina 2+ level on the 4 point Angina Scale; 2) > 2 mm horizontal or downslope ST-segment depression from resting ECG or ST-segment elevation; 3) development of new wall motion abnormalities; 4) drop in systolic SBP of > 20 mmHg below baseline despite an increase in work load; 5) complex ventricular ectopy; 6) chronotropic impairment; 7) exercise-induced left bundle branch block; 8) onset of second or third degree A-V block; and 9) severe shortness of breath, wheezing, pallor, or signs of severe peripheral circulatory insufficiency. Immediately post-exercise the subject returned to left lateral decubitus position and echocardiographic images were obtained within 30 seconds of SL-GXT termination.

Visit 2: Experimental Protocol

During the second visit subjects reported to the laboratory to perform an additional SL-GXT. The objectives of this test were to determine measured peak oxygen consumption and to serve as a supplementary screen for contraindication to participation in the study. Pre-test BP and ECG recordings were obtained. Electrocardiographic monitoring, HR and RPE were recorded each minute throughout the test and recovery. Blood pressure was measured during each 2 minute stage of exercise, at peak exercise, immediate post exercise, 1, 3, 5 and 7 minutes of recovery.

During the test, subjects breathed through a mouthpiece attached to a low-resistance breathing valve. A nose-clip was attached to the nose and expired air was analyzed for fractional oxygen (O_2), carbon dioxide (CO_2) gas concentration and expiratory minute volumes (V_E) using the Medical Graphic Cardiopulmonary Exercise Gas Analyzer CPX (Medical Graphics ™, St. Paul, MN). Exercise progressed until subject requested to stop the test or until symptoms or cardiovascular abnormalities (as outlined above) warranted termination of the test.

Visit 3: Experimental Protocol - Maximal Strength Evaluation (Experiment 1)

Maximal strength evaluations were performed on two exercises: 1) BIC incorporating the upper body, mainly the arm, using dumbbells (Sports World, Ashdod, Israel), and 2) bilateral KE utilizing the lower body, mainly thighs, using a knee extension machine (Sports World, Ashdod , Israel). The resistance apparatus utilized in this study are representative of equipment used in cardiac rehabilitation programs phases II and III.

Each testing evaluation started with a dynamic warm-up of 6 to 8 repetitions with a light weight. One-repetition maximum was determined by having the subject perform a single repetition with progressively heavier weights. Subjects started with a light weight. Upon successful completion, 2.5-10 kg were added for the next repetition. Exactly how much weight was added depended on how easy the previous repetitions were rated using the Borg scale. Subjects rested between attempts for a minimum of 3 minutes, or until HR and BP values returned to near baseline, regardless of whether the subject felt recovered sooner. Between the two different tests, i.e. KE and BIC the subject rested nearly 10 minutes. Maximal strength was defined as the maximum weight that could be lifted for one repetition through a full range of motion using good form. The test usually required 4-5 trials to complete. During each attempt the subjects were instructed to exhale while performing the concentric part of the lift. A verbal cadence was given in order to perform the maneuver for 6 seconds duration; 2 seconds for the concentric phase and 4 seconds for the eccentric phase. The KE and BIC tests were randomized to prevent an order effect.

Echocardiographic (see section entitled "Echocardiographic Measurements") and ECG measures were made throughout rest, exercise, and at the end of each minute of recovery from each trial. The principal echocardiographic images that were taken during strength tests were parasternal long- and short-axis views. Blood pressure measurements were made prior to, immediately post the lifting phase of each 1-RM trial, and during recovery.

Blood pressure measurement during maximal strength evaluation. For testing on BIC and KE, BP was measured by arm auscultation. A sphygmomanometer was positioned on the upper arm opposite the exercising arm. The bladder of the cuff was placed over the medial aspect of the upper arm. The stethoscope was positioned on the brachial artery in the antecubital region. Since BP measures are known to return to baseline values within 5-10 seconds following a lift (Wiecek et al., 1990), the cuff was inflated prior to the initiation of the lift and bled off during the lift, thus, measurement was taken immediate post the exercise (lift trial).

Visit 4: Experimental Protocol - Resistance Exercise Evaluation (Experiment 2)

For experiment 2, subjects performed repetitive resistance exercise with both BIC and KE exercises. To systematically evaluate the effect and safety of varying resistance exercise intensities on the subjects, work loads were started at a low intensity (20% of 1-RM testing) for 15 repetitions and increased progressively. Resistance exercise intensities progressed to 40% for 12 repetitions and 60% for 10 repetition based on 1-RM testing. Subjects rested between bouts for at least 3 minutes, or until HR and BP had returned to resting values and/or if the subject felt recovered. Then the next bout (next intensity) was performed. Exercise progressed until all three bouts were completed or until symptoms or cardiovascular abnormalities as outlined in "Diagnostic Graded Exercise Test" section warranted cessation of exercise. Echocardiographic, ECG and BP measures were performed during rest, throughout exercise, immediate post exercise and recovery minutes. To help avoid the Valsalva maneuver, the subjects were instructed to not hold their breath but rather to exhale at the beginning of each lift. A cadence of 6 seconds was given verbally for each repetition. One-arm biceps curl and KE exercise were randomized to prevent an order effect. Between the two different exercises, i.e. KE and BIC the subject rested about 10 minutes.

Echocardiographic (see section entitled "Echocardiographic Measurements") and ECG measures were made throughout rest, exercise, and at the end of each minute of recovery from each set. The echocardiographic images that were taken during strength tests were parasternal long- and short-axis and apical 2- and 4- chamber views.

Blood pressure measurement during resistance exercise. To evaluate BP during resistance exercise, BP measurements were made twice during each intensity set. The first measurement was taken close to the mid point of the set, while the second measurement was performed toward the final repetitions. For a specific technique description see section entitled "Blood Pressure Measurement During Maximal Strength Evaluation".

Visit 5: Experimental Protocol

Visit 5 was intended to evaluate test re-test reliability. All the procedures described in "Visit 4: Experimental Protocol" sections were repeated exactly the same during visit 5.

Data Analysis

Data were tabulated and basic descriptive statistic determination for most variables (mean ± SD) was performed. Comparisons of echocardiographic, wall motion scores, indirect BP, HR, SV and CO measurements during rest, exercise and recovery were made as follows: Experiment 1 - An analysis of variance (ANOVA) with repeated measures was used to test for differences in response to strength testing. Experiment 2 - An analysis of variance with repeated measures was used to test the differences within and between intensity bouts.

Descriptive statistics were used to report wall motion changes during exercise tests and submaximal resistance exercise bouts.

To evaluate the reliability of the procedures correlation test and paired *t*-test between the results of the 4th and 5th visits were performed.

Significant F-ratio's were evaluated by defining relevant contrasts. Alpha levels were initially set at 0.05.

CHAPTER 4
RESULTS

Descriptive Characteristics

Physical characteristics and mean descriptive data of the patients are presented in table 4-1. The subjects' ages ranged from 50 to 74 years, height and weight ranged from 155.0 - 176.0 cm and 63.0 - 97.0 kg respectively. The highest body fat percentage was 32.7% while the lowest was 20.1%. Peak aerobic capacity values (VO_{2peak}) are presented in milliliter per kilogram body weight per minute ($ml \cdot kg^{-1} \cdot min^{-1}$), with values ranging from 17.5 to 29.3 $ml \cdot kg^{-1} \cdot min^{-1}$. Ejection fraction values ranged from 31 to 48%. Clinical descriptions of the patients are summarized in table 4-2.

Values of 1-RM strength tests are also listed in table 4-1. The one-arm biceps curl (BIC) represents upper body strength involving mainly the biceps brachii, brachialis and brachioradialis muscles. The knee extension (KE) exercise depicts lower body strength, primarily thighs (quadriceps). Values for KE 1-RM ranged from 17.5 to 74.0 kg, while for BIC the weight ranged from 6.0 to 16.0 kg. The 1-RM strength of the KE test was significantly ($p \leq 0.05$) higher than that observed while performing the BIC strength test.

Clinical Symptoms

During SL-GXT six patients (40%) demonstrated down sloping ST segment depression that ranged from 1.5 to 2 mm and continued for 5 minutes into recovery. One of the patients had in addition to his ST segment depression, an inverted T wave that rotated upward during SL-GXT and the KE 1-RM test. Such ST segment changes were not observed during both 1-RM strength tests. Two patients complained of angina symptoms during the last 2 stages of the SL-GXT, which for one was coupled with a headache. Similar complaints were not expressed during strength tests.

One patient demonstrated premature ventricular and atrial contractions (PVC's and PAC's) during SL-GXT, whereas, three patients demonstrated PVC's and PAC's during recovery. Arrhythmias were not seen during KE and BIC 1-RM tests.

When performing submaximal resistance exercise bouts, only two patients demonstrated ischemic changes on the ECG. One patient had T wave changes, i.e. turning from negative to positive at 60% 1-RM KE resistance exercise, but with no ST segment depressions which was seen during SL-GXT. The other patient exhibited down sloping ST segment depression during the final repetitions of 60% 1-RM KE resistance exercise. However, the depression depth was only 0.5 mm during resistance exercise as compared to 2 mm depression during SL-GXT.

During the 60% 1-RM KE resistance exercise bout, the same two patients that reported chest pain (2+ on the 4 point Angina Scale) during SL-GXT also reported pain at the back of the neck (1 pt) and a light headache (1 pt). According to them, these signs usually appear before they get true angina pain. Nevertheless, none of them had genuine chest pain during the exercise bout, and the complaints were not followed by ischemic changes on the ECG.

Occasional arrhythmias were seen during submaximal resistance exercise. Three patients had some PVC's and PAC's during the recovery periods but not during exercise bouts. One patient had PAC's during 20% and 60% of 1-RM BIC resistance exercise work loads. Another patient had PVC's and PAC's during KE resistance exercise at 60% 1-RM. One patient had a drop of 20 mmHg (from 182 to 162 mmHg) in SBP during 60% of 1-RM KE resistance exercise.

Responses During Symptom Limited Exercise Test and Strength Tests

Hemodynamic Responses During Exercise Tests

Resting and peak exercise values for HR, systolic BP (SBP), diastolic BP (DBP) and RPP during strength tests and treadmill test are shown in table 4-3. A significant increase in HR was seen during each exercise mode compared to resting values ($p \leq 0.05$). Peak HR values during SL-GXT increased significantly ($p \leq 0.05$) compared to baseline values and was significantly higher ($p \leq 0.05$) compared to HR values attained during BIC and KE strength tests (136.4 vs. 84.0 and 84.3 beat·min^{-1} respectively). There were no significant differences ($p \geq 0.05$) in mean HR values between KE and BIC strength tests. The pattern of HR response across exercise tests is illustrated in figure 4-1.

Blood pressure measurements during the SL-GXT were performed during the final stage, however, BP during 1-RM tests were taken immediately post exercise, whereas the cuff was inflated prior to the maneuver and deflation started as the movement was completed. During the three types of exercise there were significant differences ($p \leq 0.05$) between resting and peak SBP values. Significantly higher ($p \leq 0.05$) SBP values were obtained during peak SL-GXT compared to KE and BIC strength tests. Between KE and BIC 1-RM strength tests there were no significant differences in SBP values ($p \geq 0.05$).

Significant differences ($p \leq 0.05$) between rest and peak DBP values were found only in SL-GXT and BIC tests values. Among the three different test modes, there was no significant difference ($p \geq 0.05$) in DBP values during SL-GXT compared to BIC values with a trend for higher ($p \leq 0.056$) values during SL-GXT compared to KE. Significant differences in DBP values were seen between KE and BIC strength tests ($p \leq 0.05$). Peak RPP values increased significantly ($p \leq 0.05$) from rest to exercise and were significantly higher ($p \leq 0.05$) during SL-GXT compared to KE and BIC strength tests. The pattern in BP response during the three exercise modes is shown in figure 4-2.

Values for MAP and TPR during rest and the peak exercise test modes are listed in table 4-4. Compared to rest there was a significant increase ($p \leq 0.05$) in MAP values during all test modes. During SL-GXT significantly higher peak MAP values were seen compared to both KE and BIC ($p \leq 0.05$) and between KE vs. BIC strength tests (($p \leq 0.05$). Total peripheral resistance decreased significantly from rest to exercise across all test types. During both KE and BIC strength tests, TPR values were significantly ($p \leq 0.05$) higher compared to SL-GXT. There was a trend for higher TPR value during BIC compared to a KE strength test ($p \leq 0.06$).

Echocardiographic Evaluations During Exercise Tests

Echocardiographic images for two of the patients had a lower quality but were acceptable compared to the rest of the patient group, nevertheless, echocardiographic analyses were performed on all 15 patients during strength tests and submaximal resistance exercise. Echocardiographic evaluations of left ventricular end-diastolic and systolic dimensions and left ventricular end-diastolic and systolic volumes at rest and during the three different tests modes are listed in table 4-5. Peak LVEDD during SL-GXT was significantly higher ($p \leq 0.05$) compared to resting values. However, peak LVEDD during both strength tests did not differ significantly from rest ($p \geq 0.05$). There were significantly higher ($p \leq 0.05$) LVEDD values during SL-GXT compared to both the 1-RM tests and between KE vs. BIC ($p \leq 0.05$). No significant changes ($p \geq 0.05$) from rest to exercise and between the exercise modes were observed for LVESD.

Left ventricular-end diastolic volume increased significantly ($p \leq 0.05$) from rest to SL-GXT and KE exercise only. Values during SL-GXT were significantly higher ($p \leq 0.05$) compared to KE and BIC 1-RM (183.98 vs. 171.94 and 166.01 ml respectively) and significantly higher ($p \leq 0.05$) during KE compared to BIC strength test. For LVESV values there were no significant differences ($p \geq 0.05$) across all conditions.

Changes in EF, SV, CO and SBP/LVESV ratio values from rest toward peak exercise tests are presented in table 4-6. Peak EF values during SL-GXT were significantly greater ($p \leq 0.05$) compared to the resting value (49 vs. 42% respectively) and to both KE and BIC 1-RM tests (49 vs. 42 and 43% respectively). However, there were no significant changes ($p \geq 0.05$) in EF values from rest to exercise during both strength tests.

Compared to rest, significantly higher ($p \leq 0.05$) SV values were seen during SL-GXT and a trend ($p \leq 0.07$) toward higher values were noticed during the KE test. Between the two strength tests a trend was depicted ($p \leq 0.06$) for a higher SV value during the KE strength test compared to BIC test. Significantly higher ($p \leq 0.05$) SV values were observed during SL-GXT compared to both the KE and BIC 1-RM tests.

Cardiac output changed significantly ($p \leq 0.05$) from rest to exercise demonstrating higher values during all exercise modes. Among tests, significantly higher CO values were attained during SL-GXT vs. strength tests ($p \leq 0.05$). There were no significant differences found ($p \geq 0.05$) between the strength tests

Values of SBP/LVESV ratio increased significantly ($p \leq 0.05$) from rest to exercise across test modes. During the SL-GXT values were significantly higher compared to both strength tests ($p \leq 0.05$). For BIC 1-RM test the SBP/LVESV value ratio was significantly higher ($p \leq 0.05$) compared to KE strength test.

Responses During Resistance Exercise Bouts

Submaximal resistance exercise intensities were calculated as percentages of the 1-RM test protocol. For each exercise mode, i.e., KE and BIC, the calculated intensities were 20%, 40% and 60% of the 1-RM and performed with 15, 12, and 10 repetitions respectively for each exercise bout. The mean ± SD of work load for each intensity and the RPE are shown in table 4-7. The work load ranged for KE at 20% of 1-RM from 3.5

to 15.0 kg, and ranged from 7.0 - 30.0 and 10.5 - 44 kg for 40 and 60% 1-RM respectively. For the BIC the exercise work load ranged from 1.0-4.0, 2.0-6.0 and 4.0-10.0 kg at 20, 40 and 60% of 1-RM respectively.

Responses During Knee Extension Resistance Exercise

The HR and BP responses during KE resistance exercise bouts are presented in table 4-8. The changes in the hemodynamic responses are illustrated in figures 4-3 and 4-4.

Heart rate. During exercise bouts HR increased significantly ($p \leq 0.05$) above resting values at the set's mid point (repetitions 5 through7) and at the final repetitions of the set, across intensities. As exercise bouts proceeded, significantly higher HR values were observed at the final repetitions compared to repetitions 5-7 ($p \leq 0.05$) during all work loads. Among intensities there were significant differences ($p \leq 0.05$) in the HR response as the work loads increased from 20% to 40% and 60% at the mid point of the set (85, 88, 91 $\text{beat} \cdot \text{min}^{-1}$ respectively) and towards the end of the set (92, 97 and 99 $\text{beat} \cdot \text{min}^{-1}$ respectively). Immediate post exercise HR values were significantly lower ($p \leq 0.05$) from the HR values found during the final repetitions at sets of 20 and 40% of 1-RM.

Systolic blood pressure. Due to missing data, statistical analysis on BP values during KE resistance exercise included 11 subjects. During the KE exercise SBP increased significantly ($p \leq 0.05$) above resting values at all work loads. For all intensities, SBP values during final repetitions were significantly higher ($p \leq 0.05$) than at the set's mid point (repetitions 5-7) SBP values. However, values among the sets were significantly different ($p \leq 0.05$) only between 60% 1-RM compared to 20% 1-RM (178 vs. 172 mmHg, respectively). Immediate post exercise SBP values were significantly lower ($p \leq 0.05$) compared to the final repetitions.

Diastolic blood pressure. During KE resistance exercise DBP values were significantly higher ($p \leq 0.05$) compared to rest for all exercise bouts. When comparing DBP values during repetitions 5-7 within intensities, significant differences were seen only between 20% vs. 40 and 60% of 1-RM (90 vs. 101 and 103 mmHg, respectively). Significantly higher DBP values were noted during the final repetitions compared to repetitions 5 through 7 ($p \leq 0.05$) for the 20 and 60% 1-RM work loads. There was a trend ($p \leq 0.08$) for higher DBP values at the final repetitions compared to repetitions 5-7 at 40% 1-RM. As work load intensities increased from 20% to 40% and 60% 1-RM there were significantly higher ($p \leq 0.05$) DBP values (98, 107 and 120 mmHg, respectively) at the final repetitions of the sets. Significantly lower DBP values ($p \leq 0.05$) were obtained at immediate post exercise compared to the final repetitions at all intensities.

Rate pressure product. The rate pressure product (RPP) values increased significantly during resistance exercise ($p \leq 0.05$) compared to rest. The rate pressure product (RPP) values increased significantly during exercise bouts ($p \leq 0.05$) from repetitions 5-7 toward the final repetitions of the set, in all intensities. Among intensities during repetitions 5-7 significant differences ($p \leq 0.05$) were observed between 20% vs. 40 and 60% 1-RM (126 vs. 136 and 142 $\text{mmHg} \cdot \text{min}^{-1}$, respectively), whereas, significant differences ($p \leq 0.05$) in RPP values were seen between all intensities during the final repetitions of the set. Significantly lower ($p \leq 0.05$) RPP values were observed in the immediate post exercise values compared to the final repetitions of the set for all work loads.

Mean arterial blood pressure and TPR values during rest and submaximal resistance exercise are shown in table 4-9.

Mean arterial blood pressure. There was a significant increase ($p \leq 0.05$) in MAP values from rest to exercise in all exercise bouts. Final repetitions MAP values were significantly higher ($p \leq 0.05$) compared to the set's mid point in all intensities. Among intensities during repetitions 5-7 significant differences were shown only between the 20% vs. 40 and 60% 1-RM values ($p \leq 0.05$). However, at the final repetitions there were significant differences ($p \leq 0.05$) among all the intensities (126, 135 and 141 mmHg for 20%, 40% and 60% 1-RM respectively). Immediate post exercise MAP values were significantly lower ($p \leq 0.05$) compared to final repetition values.

Total peripheral resistance. Data demonstrated no significant ($p \geq 0.05$) changes in TPR from rest to KE resistance exercise. There were no significant differences in TPR among sets ($p \geq 0.05$). Significant decreases ($p \leq 0.05$) in TPR were seen in immediate post exercise compared to the end of the set (final repetitions) under all conditions.

Responses During One-Arm Biceps Curl Resistance Exercise

Responses of HR and BP during BIC resistance exercise bouts are presented in table 4-10. Temporal changes of HR and BP during the exercise bouts are portrayed in figures 4-5 and 4-6.

Heart rate. For all intensities during BIC resistance exercise, there were significant increases in HR values ($p \leq 0.05$) from rest to repetitions 5-7. There was a further significant increase in HR values between repetitions 5-7 compared to the final repetitions ($p \leq 0.05$), for all intensities. The comparison among work loads showed significant differences ($p \leq 0.05$) between HR values for repetitions 5-7 and final repetitions for each set. Among and within all workloads there were significant differences between immediate post exercise HR values compared to final repetitions HR values ($p \leq 0.05$).

Systolic blood pressure. Statistical analyses of BP values during BIC resistance exercise were done on 13 subjects. During BIC resistance exercise SBP increased significantly ($p \leq 0.05$) from rest to exercise in all three sets. As the sets progressed a further significant increase ($p \leq 0.05$) was observed between repetitions 5-7 compared to final repetitions. Among intensities during repetitions 5-7, significant differences ($p \leq 0.05$) were noted between 20% 1-RM compared to 40 and 60% 1-RM. During the final repetitions significant differences ($p \leq 0.05$) were shown among all intensities (149, 154 and 161 mmHg for 20, 40 and 60% 1-RM respectively). Immediate post exercise SBP values were significantly lower ($p \leq 0.05$) compared to SBP values at the final repetitions of the set for all intensities.

Diastolic blood pressure. Compared to rest DBP values increased significantly ($p \leq 0.05$) during BIC resistance exercise in all exercise bouts. For 20 and 60% 1-RM work loads only significantly higher DBP values were observed during the final repetitions compared to repetitions 5-7. Significant differences ($p \leq 0.05$) among all three intensities were seen in repetitions 5-7, whereas, values differed significantly ($p \leq 0.05$) only between work loads 60% vs. 20 and 40% at the final repetitions stage. However, among workloads 20 and. 40% there was a trend towards higher DBP values at 40% ($p \leq 0.08$)

compared to 20% 1-RM during the final repetitions. The values of DBP at the end of the set were significantly higher ($p \leq 0.05$) compared to immediate post values in all intensities.

Rate pressure product. During BIC resistance exercise RPP increased significantly ($p \leq 0.05$) from repetitions 5-7 toward final repetitions of the set in all resistance exercise bouts. Among intensities, significant differences ($p \leq 0.05$) were observed in RPP values between 20% vs. 40 and 60% 1-RM for repetitions 5-7 (109 vs. 120 and 125 $mmHg \cdot min^{-1}$ respectively). Final repetitions differed significantly ($p \leq 0.05$) within all work loads (118, 132, 145 $mmHg \cdot min^{-1}$, respectively). As for immediate post exercise RPP values, there was a significant reduction ($p \leq 0.05$) compared to final repetition values.

Changes in MAP and TPR values from rest to submaximal BIC resistance exercise are shown in table 4-11.

Mean arterial pressure. Rest and exercise MAP values differed significantly ($p \leq 0.05$) during BIC resistance exercise in all intensities. Among intensities, MAP values during repetitions 5-7 were significantly different between 20% vs. 40 and 60% 1-RM. There were significant increases ($p \leq 0.05$) in MAP values from repetitions 5-7 toward the final repetitions of the set in all intensities. For the final repetitions MAP values, there were significant differences among all work loads ($p \leq 0.05$) (111, 115, and 121 mmHg, respectively). There was a significant reduction ($p \leq 0.05$) from the final repetitions MAP values compared to immediate post values for all work loads.

Total peripheral resistance. There were no significant difference ($p \geq 0.05$) in TPR values from rest to exercise across all intensities. Among all intensities there were significantly lower TPR values during immediate post exercise compared to the final repetitions of the set ($p \leq 0.05$).

Comparison between KE and BIC resistance exercise. Figures 4-7 through 4-9 demonstrate the comparison of HR, SBP, DBP, MAP and RPP, respectively between KE vs. BIC during the exercise resistance bouts. For each intensity (20%, 40% and 60% 1-RM), for both measurements (repetitions 5-7 and final repetitions) there were significant differences ($p \leq 0.05$) between KE compared to BIC in all hemodynamic variables except for DBP values at the first work load (20% 1-RM) ($p \geq 0.05$). The figures show significantly higher values for all tests during KE exercise compared to BIC ($p \leq 0.05$).

Peak values of RPP during SL-GXT, strength tests and during KE and BIC resistance exercise work loads are illustrated in figure 4-10. Peak RPP values during the KE and BIC strength tests were significantly lower ($p \leq 0.05$) during the 1-RM tests compared to all three submaximal KE work loads. Peak RPP values were significantly lower ($p \leq 0.05$) during resistance exercise compared to SL-GXT. There were significant differences ($p \leq 0.05$) among intensities for both KE and BIC demonstrating significant increase ($p \leq 0.05$) in RPP values with the increment of workloads. Comparison between peak RPP values during BIC resistance exercise vs. strength tests demonstrated significantly ($p \leq 0.05$) higher peak RPP values during 60% of 1-RM compared to both 1-RM tests.

Echocardiographic Evaluations During Knee Extension Resistance Exercise

Echocardiographic evaluations during KE resistance exercise bouts for left ventricular dimensions and volumes are presented in tables 4-12.

Left ventricular-end diastolic dimension. Significant increases ($p \leq 0.05$) in LVEDD were observed for both 40 and 60% of 1-RM work loads at the final repetitions of the set compared to rest. In addition, significantly higher ($p \leq 0.05$) LVEDD values were found at the set's mid point compared to rest during the last set (60% of 1-RM). Among intensities significantly larger ($p \leq 0.05$) LVEDD values were observed at the set's mid point during the last set (60% of 1-RM) compared to the first set (20% 1-RM). There were significant differences ($p \leq 0.05$) between the final repetitions LVEDD values compared to repetitions 5-7 values only at the second work load. There were no significant differences ($p \geq 0.05$) in final repetitions LVEDD values among intensities. There were no significant differences ($p \geq 0.05$) between immediate post exercise LVEDD values compared to the final repetitions values for both 20 and 40% of 1-RM work loads. During the last set a trend ($p \leq 0.054$) for an increase in LVEDD was found immediately post exercise compared to final repetitions.

Left ventricular-end systolic dimension. There were significantly lower ($p \leq 0.05$) LVESD values during 20% 1-RM exercise bout compared to rest. During the final set the LVESD values were significantly larger ($p \leq 0.05$) at the final repetitions compared to rest. Among intensities significant differences ($p \leq 0.05$) in LVESD values were observed between 60% vs. 20 and 40% submaximal work loads at repetitions 5 through 7. Significantly larger LVESD values were observed during intensities 40 and 60% of 1-RM compared to the first intensity at the final repetitions. Significant differences ($p \leq 0.05$) between final repetitions compared to repetitions 5-7 were observed during the second work load (40% 1-RM). There was a trend ($p \geq 0.07$) for larger LVESD values during the final repetition compared to the set's mid point during the last set. Immediate post exercise LVESD values did not differ significantly from final repetitions values ($p \geq 0.05$) across intensities. Among immediate post exercise LVESD values, significant differences ($p \leq 0.05$) were observed between 20 vs. 40 and 60% of 1-RM

Left ventricular-end diastolic volume. Significant changes ($p \leq 0.05$) in LVEDV from rest to exercise were observed during 60% 1-RM for both the set's mid point and the final repetitions of the set and during the final repetitions of the second set (40% 1-RM). Among intensities there were significant differences ($p \leq 0.05$) in LVEDV values during repetitions 5 to 7 between 60% 1-RM compared to 20 and 40%. Significant differences ($p \leq 0.05$) between final repetition vs. repetitions 5-7 were observed during the second set. During the last set there was a trend ($p \leq 0.052$) for larger LVEDV values during the final repetitions compared to mid point of the set. Significant differences ($p \leq 0.05$) were observed between 20% vs. 40 and 60% of 1-RM during the final repetitions. A trend ($p \leq 0.06$) for an increase in mean LVEDV values was found at the highest intensity (60% 1-RM) between immediate post exercise values compared to the final repetitions of the set. Among intensities, immediate post exercise LVEDV values were significantly different ($p \leq 0.05$) between 60% vs. 20 and 40% of 1-RM.

Left ventricular-end systolic volume. A significant reduction ($p \leq 0.05$) in LVESV was found during the first set when comparing rest to both the set's mid point and the final repetitions. There were significantly higher LVESV values ($p \leq 0.05$) during the final repetitions at 60% of 1-RM compared to rest. Significant differences ($p \leq 0.05$) between the final repetitions vs. repetitions 5 through 7 were seen during the second and the third sets. Among intensities there were significantly higher (p≤0.05) LVESV values at 60% 1-RM work load compared to 20% during repetitions 5-7. Significant differences (p≤0.05) in LVESV values were found across intensities during the final repetitions. There were no significant differences (p≥0.05) between immediate post exercise values compare to final repetitions across intensities. Among intensities there were significant differences (p≥0.05) in LVESV values immediately post exercise.

Values of EF, SV, CO and SBP/LVESV ratio during KE submaximal resistance exercise are illustrated in table 4-14.

Ejection fraction. During the first two sets there were no significant changes (p≥0.05) in EF values from rest to exercise for both the mid point of the set (repetitions 5-7) and the final repetitions. Significantly lower (p≤0.05) EF values were observed during repetitions 5-7 and the final repetitions of the last set (60% 1-RM) compared to rest. There was a significant (p≤0.05) reduction in EF values between the final repetitions compared to the set's mid point during the last set. Among intensities significantly lower (p≤0.05) EF values were noticed during 60% 1-RM compared to both 20 and 40% 1-RM sets for both measurements. There were no significant differences (p≥0.05) in EF values immediately post exercise compared to the final repetitions across intensities. Immediately post exercise EF values were significantly lower (p≤0.05) during the last set compared to both 20 and 40% 1-RM.

Stroke volume. There was a significant increase (p≤0.05) in SV from rest to exercise in all exercise bouts, for both measurements. Significantly higher (p≤0.05) SV values were attained during the final repetitions compared to repetitions 5-7 during the first intensity (20% 1-RM). There was a trend ($p \leq 0.06$) for increased SV values during the final repetitions compared to repetitions 5-7 at the second set (40% 1-RM). Among intensities significantly higher ($p \leq 0.05$) SV values were found during 60% 1-RM compared to 20% 1-RM at the set's mid point. Immediate post exercise SV values did not differ significantly ($p \geq 0.05$) compared to the final repetitions in all intensities. During the last set immediate post SV values were significantly higher ($p \leq 0.05$) compared to the two previous sets.

Cardiac output. Significant increases in ($p \leq 0.05$) CO values from rest to the middle of the set were observed in all three submaximal work loads (4.48, 6.18, 6.55 and 7.06 $l \cdot min^{-1}$ respectively). As exercise proceeded there were significantly higher ($p \leq 0.05$) CO values during the final repetitions compared to repetitions 5 through 7 across intensities. Among sets significantly higher ($p \leq 0.05$) CO values were achieved during 60% 1-RM compared to 20 and 40% 1-RM at repetitions 5-through 7. During the final repetitions significant differences ($p \leq 0.05$) were seen in all work loads. There were significant reductions ($p \leq 0.05$) in CO values from rest to immediate post exercise in all intensities and between the intensities.

Systolic BP/LVESV ratio. For all submaximal work loads there were significant increases ($p \leq 0.05$) in SBP/LVESV ratio values during exercise compared to rest. As exercise continued a further significant increase ($p \leq 0.05$) in SBP/LVESV values was seen compared to mid point of the set. No significant ($p \geq 0.05$) differences were depicted among intensities. Immediate post SBP/LVESV values differed significantly ($p \leq 0.05$) from values attained toward the end of the set across intensities.

Echocardiographic Evaluations During One-Arm Biceps Curl Resistance Exercise

Echocardiographic evaluation of left ventricular dimensions and volumes during BIC submaximal exercise bouts are illustrated in tables 4-13.

Left ventricular-end diastolic and systolic dimensions. Left ventricular end-diastolic dimension did not differ significantly ($p \geq 0.05$) between and within intensities. For the LVESD values there were significant reductions ($p \geq 0.05$) during exercise compared to rest in all submaximal work loads. There were no significant differences ($p \geq 0.05$) in LVESD values among intensities.

Left ventricular-end diastolic and systolic volumes. There were no significant changes ($p \geq 0.05$) in LVEDV and LVESV from rest to exercise during BIC resistance exercises for all work loads. Also among intensities there were no significant ($p \geq 0.05$) differences in LVEDV and LVESV values.

Changes in EF, SV, CO and SBP/LVESV ratio values from rest to BIC resistance exercise are presented in table 4-15.

Ejection fraction. There were no significant differences ($p \geq 0.05$) between rest to exercise in all 3 exercise bouts for all intensities. However there was a trend ($p \leq 0.058$) for lower EF values during the final set (60% 1-RM) vs. the first set (20% 1-RM).

Stroke volume. Stroke volume values increased significantly ($p \leq 0.05$) from rest to exercise across intensities. Among intensities and within intensities there were no significant differences ($p \geq 0.05$).

Cardiac output. Cardiac output increased significantly ($p \leq 0.05$) from rest to exercise across intensities. Among intensities there were significant differences ($p \leq 0.05$)

during repetitions 5-7 across intensities (5.98, 6.26 and 6.48 $l \cdot min^{-1}$ for 20, 40 and 60% 1-RM, respectively). There were significantly higher ($p \leq 0.05$) CO values during the final repetitions compared to set's mid point across intensities. Among work loads significant differences ($p \leq 0.05$) for CO values were observed during the final repetitions. Immediately post exercise CO values were significantly lower ($p \leq 0.05$) compared to final repetition values across intensities.

Systolic BP/LVESV ratio. Significant increases ($p \leq 0.05$) from rest to exercise in SBP/LVESV ratio values were observed in all three submaximal work loads. No significant differences ($p \geq 0.05$) were noticed between the final repetitions compared to the set's mid point. Immediate post exercise SBP/LVESV ratio values decreased significantly ($p \leq 0.05$) compared to values depicted at the end of the set.

Comparison between KE and BIC resistance exercise. Differences of echocardiographic evaluation between KE and BIC are illustrated in figures 4-11 through 4-13. A comparison of LVEDD values during resistance exercise (figure 4-11) demonstrated significant differences ($p \leq 0.05$) between the two exercise modes at the last set (60% 1-RM) and during the final repetitions of 40% 1-RM work load. There were significantly larger ($p \leq 0.05$) LVESD values during KE resistance exercise compared to BIC with increasing repetitions throughout the second and the third sets (figure 4-11).

Significantly larger ($p \leq 0.05$) LVEDV values were observed during KE compared to BIC resistance exercise at the final se (figure 4-12) t. During work load of 40% 1-RM significantly higher values were attained only during the last repetitions of KE exercise compared to BIC. Left ventricular ESV (figure 4-12) were significantly larger ($p \leq 0.05$)

during KE resistance exercise compared to BIC during the second and the third work loads.

Differences in EF between exercises were noted during the final repetitions of the last set in which significant decreases ($p \leq 0.05$) in EF were observed during KE resistance exercise compared to BIC (figure 4-13). Stroke volume values demonstrated significant differences between KE resistance exercise compared to BIC during the final repetitions of the last set (figure 4-13). Significantly lower ($p \leq 0.05$) SV values were found during KE resistance exercise compared to BIC at the midpoint of the first set. Significantly higher ($p \leq 0.05$) CO values were observed during KE resistance exercise bouts compared to BIC across conditions.

Wall Motion Abnormalities

In evaluating segmental wall motion during rest, 65% of the total segments (69 segments out of 105) demonstrated wall motion abnormalities (figure 4-14). Abnormal wall motion scores ranged between 1.0 (i.e. hypokinesis) to 2.0 (i.e. akinesis). During SL-GXT an additional 5 segments demonstrated new wall motion abnormalities (increase of 4.8% , i.e. 74/105). Similarly BIC 1-RM test resulted in 4.8% increase in wall motion abnormalities. Knee extension strength testing resulted in 7 additional segments with new wall motion abnormalities (6.7% increase) (figure 4-14).

Wall motion abnormalities during submaximal resistance exercise are summarized in figure 4-15. The lowest intensity KE exercise bout (20% 1-RM) did not produce any new wall motion abnormalities. The 40% 1-RM work load resulted in new wall motion abnormalities throughout the set (3/105 or 2.8% increase during repetitions 5-7; 5/105 or

4.8% increase during the final repetitions). The highest work load (60% 1-RM) elicited the greatest increase in new wall motion abnormalities (11/105 or 10.5% increase during repetitions 5-7; 13/105 or 12.4% increase during final repetitions).

The type of wall motion abnormalities during resistance exercise are presented in table 4-16. During SL-GXT 3 of the wall motion changes were from normal wall motion to hypokinesis. The other new wall motion abnormalities were akinesis (1/5) and dyskinesis (1/5). During KE 1-RM test 4 out of 7 of the new wall motion abnormalities were hypokinesis while the remaining 3 were dyskinesis. For BIC 1-RM test 3/5 were hypokinesis and the rest were dyskinesis.

During submaximal KE resistance exercise at 40% 1-RM 3 of the new wall motion abnormalities were hypokinesis and 2 progressed from hypkineasis to akinesis. During the last set (60% 1-RM) 7/13 of the new wall motion abnormalities were of the type hypkinesis, 3/13 akinesis and 3/13 dyskinesis. Only hypokinesis changes (2/2) were seen during 20% of 1-RM for BIC. The second work load of BIC (40% 1-RM) elicited hypokinesis in 2 out of 3 new wall motion abnormalities and akinesis in 1 out of 3. Finally, at the last set of BIC (60% 1-RM) 4/11 of the new wall motion abnormalities were of the type hypkinesis, 5/11 akinesis and 2/11 dyskinesis

Correlation Between Visit 4 and Visit 5

Correlation between Visit 4 and Visit 5 variables are summarized in table 4-17 and table 4-18, demonstrating high correlation values. The *t*-test for HR and BP variables demonstrated 12% significant differences ($p \leq 0.05$). Most of the significant differences were observed for DBP values during BIC resistance exercise, demonstrating pattern of lower values during Visit 5. The *t*-test for echocardiographic data showed 9% significant difference ($p \leq 0.05$) with no specific pattern.

Table 4-1. Descriptive data of the study participants (mean ± SD).

Variable	Values
N	15
Age (yrs)	65.0 ± 6.5
Height (cm)	165.8 ± 5.3
Weight (kg)	73.9 ± 9.2
Body fat (%)	27.3 ± 4.0
EF (%)	42.1 ± 5.8
VO_{2peak} ($ml \cdot kg^{-1} \cdot min^{-1}$)	21.48 ± 4.3
METs	6.1 ± 1.2
1-RM knee extension (kg)	45.6 ± 14.2
1-RM one-arm biceps curl (kg)	10.1 ± 2.3

VO_{2peak} - Peak oxygen consumption
MET - Metabolic equivalent
1-RM - One Repetition Maximum
EF - Ejection Fraction

Table 4-2. Clinical characteristics of the subjects.

Patient	Event	Number of vessels
1	Ant. MI	3
2	Inf. MI, CABG	2
3	Ant. MI	1
4	Inf. MI x 2	2
5	Ant.-Inf. MI, CABG	3
6	Post. MI, CABG	3
7	Ant. MI x 2, CABG x 2	3
8	Ant.-Inf. MI, CABG	2
9	Ant. MI, PTCA x2, CABG	3
10	AP, PTCA	1
11	Ant. MI x 2, PTCA x 2, CABG	3
12	Ant. MI	1
13	CABG	3
14	Inf.-Post. MI, PTCA + stent x 2	3
15	Ant. MI, PTCA	2

MI - Myocardial Infarction
Ant. - Anterior
Inf. - Inferior
Post. - Posterior
CABG - Coronary Artery Bypass Graft
PTCA - Precutaneous Transluminal Coronary Angioplasty
AP - Angina Pectoris

Table 4-3. Heart rate and blood pressure values at rest, peak strength and treadmill symptom-limited graded exercise testing (mean ± SD).

Variable	Rest	SL-GXT	1-RM KE	1-RM BIC
Heart rate (beat·min^{-1})	67.9 ± 14.8	$136.4 \pm 13.4^{\# *}$	$84.3 \pm 15.7^{\#}$	$84.0 \pm 13.9^{\#}$
Systolic pressure (mmHg)	127.8 ± 16.8	$185.9 \pm 30.9^{\# *}$	$141.7 \pm 26.3^{a \#}$	$145.1 \pm 19.3^{a \#}$
Diastolic pressure (mmHg)	76.1 ± 9.7	$85.3 \pm 12.5^{\#}$	80.0 ± 11.9^{a}	$85.5 \pm 12.2^{a \# \wedge}$
Rate pressure product^{b} (mmHg·min^{-1})	85.9 ± 24.1	$254.4 \pm 54.7^{*\#}$	$120.2 \pm 33.5^{\#}$	$122.2 \pm 27.6^{\#}$

SL-GXT - Symptom-Limited Graded Exercise Test, 1-RM - One Repetition Maximum, KE - Knee Extension, BIC - One-Arm Biceps Curl,

[a] Measurement taken immediate post exercise

[b] Peak heart rate x peak systolic pressure x 10^{-2}

[#] ($p \leq 0.05$) Exercise vs. rest

[*] ($p \leq 0.05$) SL-GXT vs. 1-RM test for both KE and BIC

[^] ($p \leq 0.05$) BIC vs. KE

Table 4-4. Comparison between peak mean arterial pressure and total peripheral resistance values at rest, peak strength and graded exercise testing (mean ± SD).

Variable	Rest	SL-GXT	1-RM KE	1-RM BIC
MAP (mmHg)	92.2 ± 13.6	118.5± $^{\# *}$ 15.5	100.4 ± $^{\#}$ 14.7	105.1 ± $^{\#\wedge}$ 11.4
TPR (mmHg·L^{-1}·min^{-1})	20.2 ± 4.5	10.2 ± $^{\# *}$ 2.6	15.8 ± $^{\#}$ 4.7	17.3 ± $^{\#}$ 5.6

SL-GXT - Symptom-Limited Graded Exercise Test
1-RM - One Repetition Maximum
KE - Knee Extension
BIC - One-Arm Biceps Curl,
MAP - Mean Arterial Pressure
TPR - Total Peripheral Resistance
$^{\#}$ ($p \leq 0.05$) Exercise vs. rest
* ($p \leq 0.05$) SL-GXT vs. 1-RM test for both KE and BIC
$^{\wedge}$ ($p \leq 0.05$) BIC vs. KE

Table 4-5. Left ventricular end-diastolic and systolic dimensions and volumes at rest, peak strength and treadmill symptom-limited graded exercise testing (mean ± SD).

Variable	Rest	SL-GXT	1-RM KE	1-RM BIC
LVEDD (cm)	5.8 ± 0.5	6.0 ± 0.6$^{\#*}$	5.9 ± 0.6$^{\wedge}$	5.6 ± 0.6
LVESD (cm)	4.5 ± 0.5	4.5 ± 0.6	4.4 ± 0.6	4.4 ± 0.6
LVEDV (ml)	165.8 ± 34.4	183.9 ± 43.6$^{\#*}$	171.9 ± 40.4$^{\#\wedge}$	166.0 ± 37.5
LVESV (ml)	92.7 ± 26.0	92.9 ± 30.3	91.7 ± 28.3	89.5 ± 28.3

SL-GXT - Symptom-Limited Graded Exercise Test
1-RM - One Repetition Maximum
KE - Knee Extension
BIC - One-Arm Biceps Curl
LVEDD - Left Ventricular End Diastolic Dimension
LVESD - Left Ventricular End Systolic Dimension
LVEDV - Left Ventricular End Diastolic Volume
LVESV - Left Ventricular End Systolic Volume,
[#] (p≤0.05) Exercise vs. rest
[*] (p≤0.05) SL-GXT vs. 1-RM test for both KE and BIC
[^] (p≤0.05) KE vs. BIC

Table 4-6. Ejection fraction, stroke volume, cardiac output and systolic blood pressure to left ventricular-end systolic volume ratio values at rest, peak strength and graded exercise testing (mean ± SD).

Variable	Rest	SL-GXT	1-RM KE	1-RM BIC
EF (%)	42.1 ± 5.8	$49.3 \pm 5.1^{\#*}$	42.1 ± 7.1	42.9 ± 7.2
SV (ml)	73.1 ± 12.9	$91.0 \pm 16.7^{\#*}$	80.2 ± 15.1	76.5 ± 12.7
CO ($l \cdot min^{-1}$)	4.5 ± 1.2	$12.2 \pm 2.8^{\#*}$	$6.7 \pm 1.5^{\#}$	$6.5 \pm 1.6^{\#}$
SBP/LVESV	1.5 ± 0.4	$2.2 \pm 0.7^{\#*}$	$1.7 \pm 0.5^{\#}$	$1.8 \pm 0.6^{\#\wedge}$

SL-GXT - Symptom-Limited Graded Exercise Test
1-RM - One Repetition Maximum
KE - Knee Extension
BIC - One-Arm Biceps Curl
EF - Ejection Fraction
SV - Stroke volume
CO - Cardiac Output
SBP/LVESV - Systolic Blood Pressure to Left Ventricular End Systolic Volume ratio
$^{\#}$ ($p \leq 0.05$) Exercise vs. rest
* ($p \leq 0.05$) SL-GXT vs. 1-RM test for both KE and BIC
$^{\wedge}$ ($p \leq 0.05$) BIC vs. KE

Table 4-7. Work loads and rating of perceived exertion for the different intensity bouts (mean ± SD).

Variable	20% 1-RM	40% 1-RM	60% 1-RM
KE (kg)	9.1 ± 2.9	18.3 ± 5.7	27.3 ± 8.4
RPE	11.7 ± 1.5	14.5 ± 1.9	15.6 ± 2.1
BIC (kg)	2.0 ± 0.6	3.9 ± 0.9	6.0 ± 1.3
RPE	9.9 ± 2.6	12.3 ± 1.8	13.9 ± 1.7

1-RM - One Repetition Maximum
KE - Knee Extension
BIC - One-Arm Biceps Curl
RPE - Rate of Perceived Exertion

Table 4-8. Heart rate and blood pressure responses during knee extension resistance exercise (mean ± SD).

		20% 1-RM			40% 1-RM			60% 1-RM		
Variable	Rest	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP
HR (beat·min^{-1}) (n=15)	69.6 ± 15.6	84.8 ±$^{\bullet *}$ 17.5	92.4 ±$^{\bullet * \#}$ 19.5	90.5 ±$^{* \upsilon}$ 19.6	87.8 ±$^{\bullet *}$ 18.4	96.7 ±$^{\bullet * \#}$ 19.7	94.3 ±$^{* \upsilon}$ 18.7	91.4 ±$^{\bullet *}$ 17.8	98.9 ±$^{\bullet * \#}$ 20.8	97.7 ±* 21.2
SBP (mmHg) (n=11)	132.4 ± 21.5	152.5 ±$^{\bullet}$ 16.8	171.8 ±$^{\bullet \#}$ 20.4	152.6 ±$^{\upsilon}$ 16.3	153.6 ±$^{\bullet}$ 22.0	173.4 ±$^{\bullet \#}$ 19.3	153.6 ±$^{\upsilon}$ 25.5	155.4 ±$^{\bullet}$ 24.6	177.9 ±$^{\bullet \Psi \#}$ 23.8	153.5 ±$^{\upsilon}$ 26.7
DBP (mmHg) (n=11)	76.6 ± 11.9	90.0 ±$^{\bullet +}$ 11.9	98.1 ±$^{\bullet * \#}$ 17.7	74.6 ±$^{\upsilon}$ 11.2	100.7 ±$^{\bullet}$ 19.5	106.9 ±$^{\bullet *}$ 21.4	73.8 ±$^{\upsilon}$ 13.6	102.7 ± • 14.2	120.0 ±$^{\bullet * \#}$ 18.4	77.3±$^{\upsilon}$ 10.9
RPP (mmHg·min^{-1}) (n=11)	86.1 ± 19.8	126.3 ±$^{\bullet +}$ 32.1	160.2 ±$^{\bullet * \#}$ 41.9	138.9 ±$^{* \upsilon}$ 37.5	135.5 ±$^{\bullet}$ 37.9	169.4 ±$^{\bullet * \#}$ 42.7	149.4 ±$^{* \upsilon}$ 44.0	141.7 ±$^{\bullet}$ 30.5	179.8 ±$^{\bullet * \#}$ 45.1	154.6 ±$^{* \upsilon}$ 49.2

1-RM - One Repetition Maximum, Reps - Repetitions, IP - Immediate Post, RPP - Rate Pressure Product (HR·SBP·10^{-2})

$^{\bullet}$ (p≤0.05) Exercise vs. rest

* (p≤0.05) Difference between intensities

$^{\#}$ (p≤0.05) Final repetitions vs. repetitions 5-7

$^{+}$ (p≤0.05) Difference between 20% vs. 40 and 60% of 1-RM

$^{\Psi}$ (p≤0.05) Difference between 60% vs.20% 1-RM

$^{\upsilon}$ Immediate post exercise vs. final repetitions

Table 4-9. Mean arterial pressure and total peripheral resistance responses during knee extension resistance exercise (mean ± SD).

		20% 1-RM			40% 1-RM			60% 1-RM		
Variable	Rest	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP
MAP (mmHg) (n=11)	94.9 ± 2.6	111.5 ±$^{\bullet +}$ 9.9	124.4 ±$^{\bullet * \#}$ 16.8	99.5±$^{\upsilon}$ 11.4	118.6 ±$^{\bullet}$ 17.8	130.9 ±$^{\bullet * \#}$ 19.3	100.5 ±$^{\upsilon}$ 14.9	119.3 ±$^{\bullet}$ 15.3	141.9±$^{\bullet * \#}$ 20.5	102.1 ±$^{\upsilon}$ 14.1
TPR ($mmHg \cdot L^{-1} \cdot min^{-1}$) (n=11)	19.5 ± 5.3	18.7 ± 6.5	18.5 ± 6.0	13.8 ±$^{\upsilon}$ 4.3	18.9 ± 7.5	18.1 ± 6.9	13.5 ±$^{\upsilon}$ 5.2	18.0 ± 6.7	18.98± 7.4	12.8 ±$^{\upsilon}$ 4.9

1-RM - One Repetition Maximum
Reps - Repetitions
IP - Immediate Post,
MAP - Mean Arterial Pressure
TPR - Total Peripheral Resistance
$^{\bullet}$ (p≤0.05) Exercise vs. rest
* (p≤0.05) Difference between intensities
$^{\#}$ (p≤0.05) Final repetitions vs. repetitions 5-7
$^{+}$ (p≤0.05) Difference between 20% vs. 40 and 60% of 1-RM
$^{\upsilon}$ Immediate post exercise vs. final repetitions

Table 4-10. Heart rate and blood pressure responses during one-arm biceps curl resistance exercise (mean ± SD).

		20% 1-RM			40% 1-RM			60% 1-RM		
Variable	Rest	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP
HR (beat·min^{-1}) (n=15)	71.1 ± 15.3	77.6 ±$^{\bullet *}$ 14.6	79.7 ±$^{\#\bullet *}$ 15.5	77.2 ±$^{\upsilon *}$ 15.9	81.8 ±$^{\bullet *}$ 15.1	84.1 ±$^{\#\bullet *}$ 16.2	82.6 ±$^{\upsilon *}$ 16.9	84.2 ±$^{\bullet *}$ 16.1	88.6 ±$^{\#\bullet *}$ 17.0	86.8 ±$^{\upsilon *}$ 17.9
SBP (mmHg) (n=13)	124.0 ± 17.2	142.0 ±$^{\bullet +}$ 17.7	149.2 ±$^{\#\bullet *}$ 18.9	132.8 ±$^{\upsilon}$ 22.3	147.2 ±$^{\bullet}$ 15.7	154.7 ±$^{\#\bullet *}$ 20.0	127.8 ±$^{\upsilon}$ 18.8	148.5 ±$^{\bullet}$ 16.1	161.2 ±$^{\#\bullet *}$ 17.1	132.1 ±$^{\upsilon}$ 21.9
DBP (mmHg) (n=13)	74.1 ± 9.5	88.1 ±$^{\bullet *}$ 8.8	93.5 ±$^{\bullet *}$ 12.7	77.8 ±$^{\upsilon}$ 8.9	94.2 ±$^{\bullet *}$ 11.5	96.1 ±$^{\bullet *}$ 13.3	75.0 ±$^{\upsilon}$ 8.5	98.5 ±$^{\bullet *}$ 9.4	103.0 ±$^{\#\bullet \Phi}$ 13.3	76.8 ±$^{\upsilon}$ 11.3
RPP (mmHg·min^{-1}) (n=13)	86.0 ± 19.9	109.8 ±$^{\bullet +}$ 21.7	118.8 ±$^{\#\bullet *}$ 29.4	101.4 ±$^{\upsilon *}$ 31.0	120.4 ±$^{\bullet}$ 25.0	132.1 ±$^{\#\bullet *}$ 32.3	107.2 ±$^{\upsilon *}$ 30.7	125.4 ±$^{\bullet}$ 27.4	145.8 ±$^{\#\bullet *}$ 34.5	117.0 ±$^{\upsilon *}$ 40.3

1-RM - One Repetition Maximum, Reps - Repetitions, IP - Immediate Post, HR - Heart Rate, RPP - Rate Pressure Product (HR·SBP·10^{-2})

$^{\bullet}$ ($p \leq 0.05$) Exercise vs. rest

* ($p \leq 0.05$) Difference between intensities

$^{\#}$ ($p \leq 0.05$) Final repetitions vs. repetitions 5-7

$^{+}$ ($p \leq 0.05$) Difference between 20% vs. 40 and 60% of 1-RM

$^{\Phi}$ ($p \leq 0.05$) Difference between 60% vs.20 and 40% 1-RM

$^{\upsilon}$ Immediate post exercise vs. final repetitions

Table 4-11. Mean arterial pressure and total peripheral resistance responses during one-arm biceps curl resistance exercise (mean ± SD).

		20% 1-RM			40% 1-RM			60% 1-RM		
Variable	Rest	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP
MAP (mmHg) (n=13)	90.9 ± 10.6	105.9 ±$^{\bullet +}$ 9.4	111.7 ±$^{\bullet * \#}$ 13.0	94.2 ±$^{\upsilon}$ 11.1	112.4 ±$^{\bullet}$ 11.5	115.8 ±$^{\bullet * \#}$ 14.9	92.8 ±$^{\upsilon}$ 10.1	115.7 ±$^{\bullet}$ 11.2	121.4 ±$^{\bullet * \#}$ 13.3	94.0 ±$^{\upsilon}$ 14.4
TPR ($mmHg \cdot L^{-1} \cdot min^{-1}$) (n=13)	19.5 ± 5.3	18.8 ± 5.7	19.3 ± 6.6	15.8 ±$^{\upsilon}$ 5.3	18.9 ± 6.1	18.9 ± 6.8	15.4 ±$^{\upsilon}$ 5.1	19.0 ± 6.1	19.0 ± 6.2	14.5 ±$^{\upsilon}$ 4.6

1-RM - One Repetition Maximum
Reps - Repetitions
IP - Immediate Post
MAP - Mean Arterial Pressure
TPR - Total Peripheral Resistance
$^{\bullet}$ ($p \leq 0.05$) Exercise vs. rest
* ($p \leq 0.05$) Difference between intensities
$^{\#}$ ($p \leq 0.05$) Final repetitions vs. repetitions 5-7
$^{+}$ ($p \leq 0.05$) Difference between 20% vs. 40 and 60% of 1-RM
$^{\upsilon}$ Immediate post exercise vs. final repetitions

Table 4-12. Changes in left ventricular end diastolic and systolic dimensions and volumes during knee extension resistance exercise (mean ± SD).

		20% 1-RM			40% 1-RM			60% 1-RM		
Variable	Rest	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP
LVEDD (cm)	5.7 ± 0.	5.7 ± 0.5	5.7 ± 0.5	5.7 ± 0.5	5.7 ± 0.5	5.8 ±$^{\bullet \#}$ 0.6	5.8 ± 0.6	5.8 ±$^{\bullet \Psi}$ 0.5	5.8 ±$^{\bullet}$ 0.6	5.8 ± 0.6
LVESD (cm)	4.5 ± 0.5	4.4 ±$^{\bullet}$ 0.5	4.4 ±$^{\bullet +}$ 0.5	4.4 ±$^{+}$ 0.5	4.5 ± 0.6	4.6 ±$^{\#}$ 0.5	4.6 ± 0.6	4.5 ±$^{\Phi}$ 0.6	4.6 ±$^{\bullet}$ 0.6	4.6 ± 0.6
LVEDV (ml)	165.7± 34.3	165.4 ± 36.3	167.2 ±$^{\bullet +}$ 36.8	167.6 ± 36.6	168.2 ± 38.5	171.9 ±$^{\bullet \#}$ 40.2	171.7 ± 40.2	171.8 ±$^{\bullet \Phi}$ 39.2	173.8 ±$^{\bullet}$ 41.2	175.1 ±$^{\Phi}$ 41.5
LVESV (ml)	92.6± 26.0	90.3 ±$^{\bullet}$ 27.8	90.1 ±$^{\bullet *}$ 28.4	89.8 ±* 28.6	91.4 ± 29.2	93.3 ±$^{* \#}$ 29.1	93.2 ±* 30.4	93.8 ±$^{\Psi}$ 30.0	95.3 ±$^{\bullet * \#}$ 31.0	96.1 ±* 32.4

1-RM - One Repetition Maximum, Reps - Repetitions, IP - Immediate Post, LVEDD - Left Ventricular End Diastolic Dimension, LVESD - Left Ventricular End Systolic Dimension, LVEDV - Left Ventricular End Diastolic Volume, LVESD - Left Ventricular End Systolic Volume,

$^{\bullet}$ ($p \leq 0.05$) Exercise vs. rest

* ($p \leq 0.05$) Difference between intensities

$^{\#}$ ($p \leq 0.05$) Final repetitions vs. repetitions 5-7

$^{\Psi}$ ($p \leq 0.05$) Difference between 60% vs.20% 1-RM

$^{+}$ ($p \leq 0.05$) Difference between 20% vs. 40 and 60% of 1-RM

$^{\Phi}$ ($p \leq 0.05$) Difference between 60% vs. 20 and 40% of 1-RM

$^{\upsilon}$ Immediate post exercise vs. final repetitions

Table 4-13. Changes in left ventricular end diastolic and systolic dimensions and volumes during one-arm biceps curl resistance exercise (mean ± SD).

		20% 1-RM			40% 1-RM			60% 1-RM		
Variable	Rest	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP
LVEDD (cm)	5.7 ± 0.5	5.7 ± 0.5	5.7 ± 0.6	5.8 ± 0.6	5.7 ± 0.5	5.7 ± 0.5	5.7 ± 0.5	5.7 ± 0.5	5.8 ± 0.6	5.8 ± 0.6
LVESD (cm)	4.4 ± 0.5	4.4 ± • 0.6	4.4 ± • 0.6	4.3 ± 0.6	4.4 ± • 0.6	4.4± • 0.6	4.4 ± 0.6	4.4 ± • 0.6	4.4 ± • 0.6	4.4 ± 0.6
LVEDV (ml)	165.7 ± 34.3	167.4 ± 38.9	168.4 ± 39.5	169.2 ± 39.6	166.6± 38.7	169.2 ± 38.1	167.9 ± 39.1	167.8 ± 39.1	169.3 ± 40.3	170.2 ± 40.8
LVESV (ml)	92.6 ± 26.0	90.4 ± 30.1	90.2 ± 30.9	89.7 ± 31.4	90.1± 29.8	90.4 ± 30.6	91.4 ± 31.0	90.9 ± 29.9	91.5 ± 31.0	92.5 ± 31.8

1-RM - One Repetition Maximum, Reps - Repetitions, IP - Immediate Post, LVEDD - Left Ventricular End Diastolic Dimension, LVESD - Left Ventricular End Systolic Dimension, LVEDV - Left Ventricular End Diastolic Volume, LVESD - Left Ventricular End Systolic Volume,

• ($p \leq 0.05$) Exercise vs. rest

Table 4-14. Changes in ejection fraction, stroke volume, cardiac output and systolic blood pressure to left ventricular-end systolic volume ratio during knee extension resistance exercise (mean ± SD).

		20% 1-RM			40% 1-RM			60% 1-RM		
Variable	Rest	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP
EF (%)	42.7 ± 6.7	42.8 ± 7.1	42.4± 7.8	42.6 ± 8.0	42.9 ± 7.3	42.4 ± 7.6	42.5± 7.7	41.1 ± $^{\bullet\Phi}$ 7.6	40.0 ± $^{\bullet\Phi\#}$ 8.7	40.4 ± $^{\Phi}$ 8.9
SV (ml)	73.1 ± 12.9	75.9 ± $^{\bullet}$ 12.4	77.6 ± $^{\bullet\#}$ 12.3	77.7± 11.9	76.7 ± $^{\bullet}$ 13.1	78.5 ± $^{\bullet}$ 14.8	77.8± 13.8	78.3 ± $^{\bullet\Psi}$ 11.9	78.8 ± $^{\bullet}$ 12.8	79.3± $^{\Psi}$ 12.3
CO (l·min^{-1})	4.8 ± 1.2	6.1± $^{\bullet}$ 1.5	6.9 ± $^{\bullet * \#}$ 1.8	6.9 ± $^{\bullet}$ 1.8	6.5 ± $^{\bullet}$ 1.8	7.6± $^{\bullet * \#}$ 2.1	7.3 ± $^{* \upsilon}$ 1.9	7.0 ± $^{\bullet\Phi}$ 2.0	7.7 ± $^{\bullet * \#}$ 2.2	7.7 ± $^{* \upsilon}$ 2.2
SBP/LVESV (n=11)	1.5 ± 0.4	1.8 ± $^{\bullet}$ 0.6	2.1 ± $^{\bullet\#}$ 0.7	1.8 ± $^{\upsilon}$ 0.5	1.8 ± $^{\bullet}$ 0.6	2.1 ± $^{\bullet\#}$ 0.6	1.8 ± $^{\upsilon}$ 0.5	1.8 ± $^{\bullet}$ 0.6	2.1 ± $^{\bullet\#}$ 0.7	1.8 ± $^{\upsilon}$ 0.6

1-RM - One Repetition Maximum, Reps - Repetitions, IP - Immediate Post, EF - Ejection Fraction, SV - Stroke Volume, CO - Cardiac Output, SBP/LVESV - Systolic Blood Pressure to Left Ventricular End Systolic Volume ratio

$^{\bullet}$ ($p \leq 0.05$) Exercise vs. rest

* ($p \leq 0.05$) Difference between intensities

$^{\Psi}$ ($p \leq 0.05$) Difference between 60% vs.20% 1-RM

$^{\Phi}$ ($p \leq 0.05$) Difference between 60% vs. 20 and 40% of 1-RM

$^{\#}$ ($p \leq 0.05$) Final repetitions vs. repetitions 5-7

$^{\upsilon}$ Immediate post exercise vs. final repetitions

Table 4-15. Changes in ejection fraction, stroke volume, cardiac output and systolic blood pressure to left ventricular-end systolic volume ratio during one-arm biceps curl resistance exercise (mean ± SD).

		20% 1-RM			40% 1-RM			60% 1-RM		
Variable	Rest	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP	Reps. 5-7	Final Reps	IP
EF (%)	42.7 ± 6.7	42.8 ± 7.4	42.6 ± 8.1	42.7 ± 8.7	42.6 ± 7.4	41.4 ± 8.1	41.9 ± 8.4	41.4 ± 7.7	41.2 ± 8.3	41.4 ± 8.2
SV (ml)	73.1 ± 12.9	77.5 ± $^{\bullet}$ 12.8	78.1 ± $^{\bullet}$ 12.4	79.5 ± 12.2	76.5 ± $^{\bullet}$ 12.4	76.9 ± $^{\bullet}$ 11.9	76.4 ± 12.3	76.9 ± $^{\bullet}$ 12.2	77.8 ± $^{\bullet}$ 12.8	77.7 ± 12.1
CO ($l \cdot min^{-1}$)	4.8 ± 1.2	5.9 ± $^{*\bullet}$ 1.4	6.2 ± $^{*\bullet\#}$ 1.5	6.1 ± 1.6	6.2 ± $^{*\bullet}$ 1.5	6.4 ± $^{*\bullet\#}$ 1.5	6.2 ± 1.6	6.4 ± $^{*\bullet}$ 1.0	6.8 ± $^{*\bullet\#}$ 1.6	6.7 ± $^{\Phi}$ 1.7
SBP/LVESV (n=13)	1.4 ± 0.3	1.7 ± $^{\bullet}$ 0.5	1.8 ± $^{\bullet}$ 0.6	1.6 ± $^{\upsilon}$ 0.5	1.7 ± $^{\bullet}$ 0.5	1.8 ± $^{\bullet}$ 0.6	1.5 ± $^{\upsilon}$ 0.5	1.7 ± $^{\bullet}$ 0.5	1.9 ± $^{\bullet\#}$ 0.8	1.5 ± $^{\upsilon}$ 0.5

1-RM - One Repetition Maximum, Reps - Repetitions, IP - Immediate Post, EF - Ejection Fraction, SV - Stroke Volume, CO - Cardiac Output, SBP/LVESV - Systolic Blood Pressure to Left Ventricular End Systolic Volume ratio

$^{\bullet}$ ($p \leq 0.05$) Exercise vs. rest

* ($p \leq 0.05$) Difference between intensities

$^{\Phi}$ ($p \leq 0.05$) Difference between 60% vs. 20 and 40% of 1-RM

$^{\#}$ ($p \leq 0.05$) Final repetitions vs. repetitions 5-7

$^{\upsilon}$ Immediate post exercise vs. final repetitions

Table 4-16. Prevalence of wall motion abnormalities induced by exercise

Exercise		Hypokinesis	Akinesis	Dyskinesis
SL-GXT		3/5	1/5	1/5
KE 1-RM		4/7	……..	3/7
BIC 1-RM		3/5	…….	2/5
KE	20% 1-RM	…….	…….	…….
	40% 1-RM	3/5	2/5	…….
	60% 1-RM	7/13	3/13	3/13
BIC RM	20% 1-	2/2	…….	…….
	40% 1-RM	2/3	1/3	…….
	60% 1-RM	4/11	5/11	2/11

SL-GXT - Symptom Limited Graded Exercise Test
1-RM - One Repetition Maximum
KE - Knee Extension
BIC - One Arm Biceps Curl

Table 4-17. Correlation values of Visit 4 and Visit 5 hemodynamic responses and echocardiographic variables during knee extension resistance exercise (n = 11).

Variable	r
Heart rate (beat·min^{-1})	0.94
Systolic blood pressure (mmHg)	0.78
Diastolic blood pressure (mmHg)	0.79
Left ventricular-end diastolic dimension (cm)	0.97
Left ventricular-end systolic dimension (cm)	0.98
Left ventricular-end diastolic volume (ml)	0.97
Left ventricular-end systolic volume (ml)	0.98
Ejection fraction (%)	0.92
Stroke volume (ml)	0.91
Cardiac output (l·min^{-1})	0.87

All correlation were set at most at $p \leq 0.05$

Table 4-18. Correlation values of Visit 4 and Visit 5 hemodynamic responses and echocardiographic variables during one-arm biceps curl resistance exercise (n = 13).

Variable	r
Heart rate (beat·min^{-1})	0.92
Systolic blood pressure (mmHg)	0.79
Diastolic blood pressure (mmHg)	0.76
Left ventricular-end diastolic dimension (cm)	0.98
Left ventricular-end systolic dimension (cm)	0.98
Left ventricular-end diastolic volume (ml)	0.95
Left ventricular-end systolic volume (ml)	0.98
Ejection fraction (%)	0.97
Stroke volume (ml)	0.88
Cardiac output (l·min^{-1})	0.81

All correlation were set at most at $p \leq 0.05$

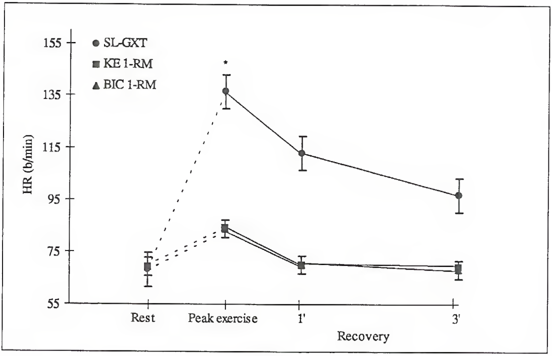


Figure 4-1. Peak heart rate (HR) values during symptom-limited exercise test (SL-GXT), knee extension (KE) and one-arm biceps curl (BIC) strength tests.
* $p \leq 0.05$ SL-GXT vs. KE and BIC

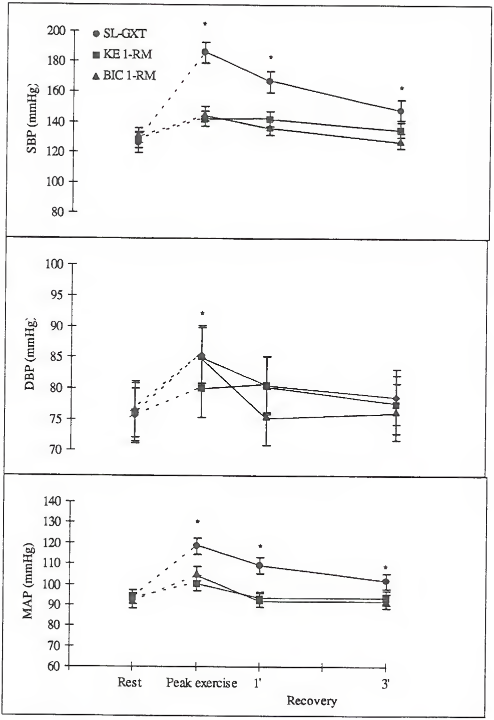


Figure 4-2. Peak systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) during symptom-limited exercise test (SL-GXT), knee extension (KE) and one-arm biceps curl (BIC) strength tests.
* p≤0.05 SL-GXT vs. KE and BIC

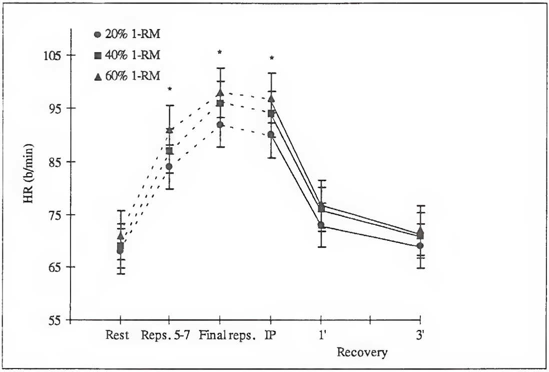


Figure 4-3. Heart rate (HR) response at rest, knee extension resistance exercise and recovery during 20, 40 and 60% of 1-RM. (mean ± SE)
* $p \leq 0.05$ between intensities

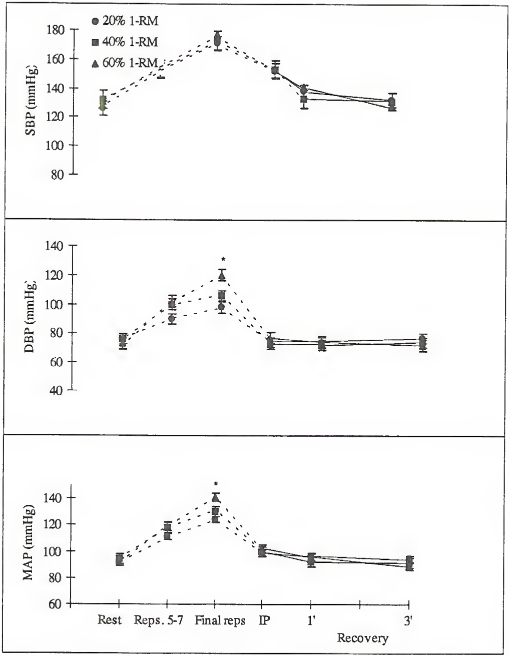


Figure 4-4. Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) responses at rest, knee extension resistance exercise and recovery during 20, 40 and 60% of 1-RM. (mean ± SE)
* p≤0.05 between intensities

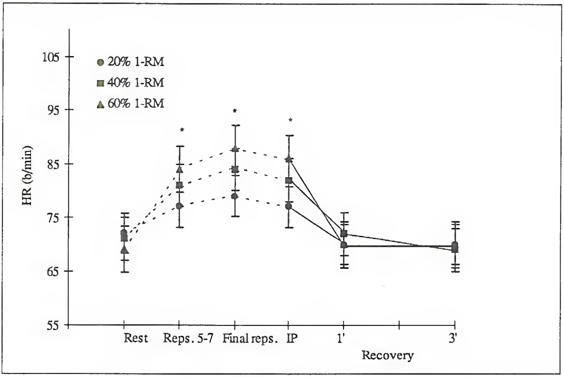


Figure 4-5. Heart rate (HR) response at rest, one-arm biceps curl resistance exercise and recovery during 20, 40 and 60% of 1-RM. (mean ± SE)
* $p \leq 0.05$ between intensities

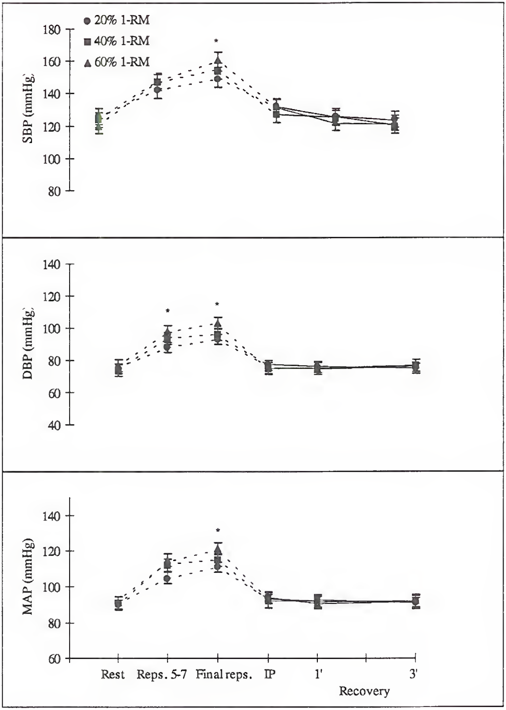


Figure 4-6. Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) response at rest, one-arm biceps resistance exercise and recovery during 20, 40 and 60% of 1-RM. (mean ± SE)
* $p \leq 0.05$ between intensities

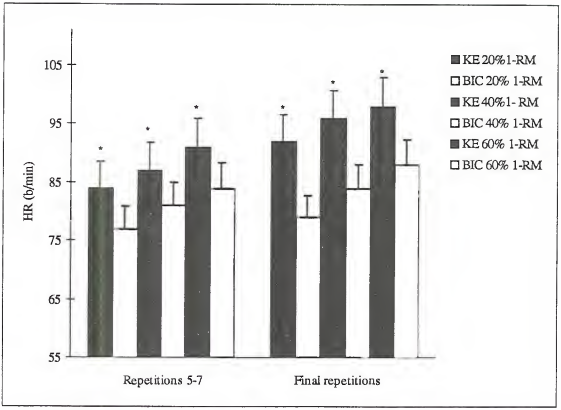


Figure 4-7. Comparison of heart rate (HR) response between knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different work load levels. (mean ± SE)
* $p \leq 0.05$ KE vs. BIC

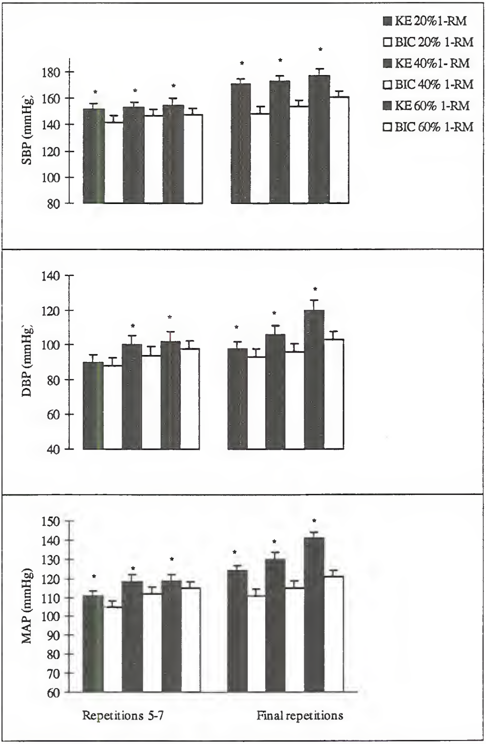


Figure 4-8. Comparison of systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) responses between knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different work load levels.
(mean ± SE)
*$p \leq 0.05$ KE vs. BIC

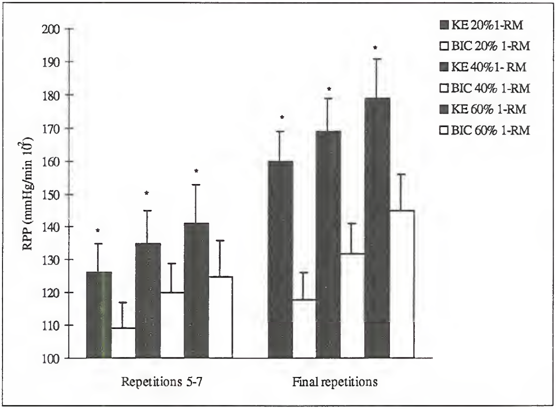


Figure 4-9. Comparison of rate pressure products (RPP) response between knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different work load levels. (mean ± SE)
* $p \leq 0.05$ KE vs. BIC

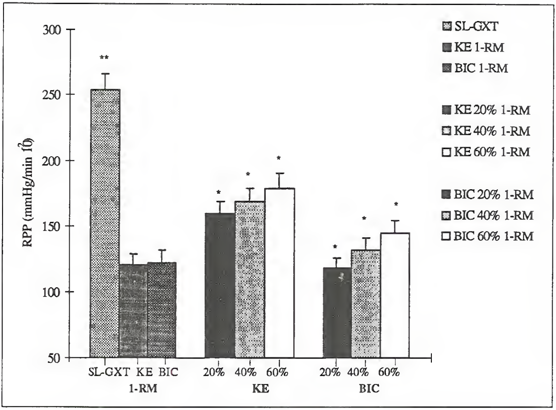


Figure 4-10. Comparison between peak rate pressure product (RPP) values during symptom-limited exercise test (SL-GXT), knee extension (KE) and one-arm biceps curl (BIC) strength tests and resistance exercises. (mean ± SE)
* $p \leq 0.05$ between intensities
** $p \leq 0.05$ SL-GXT vs. KE and BIC

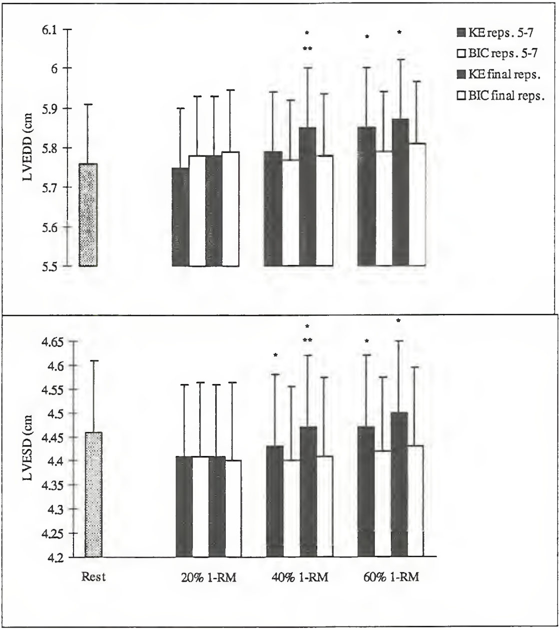


Figure 4-11. Changes in left ventricular end diastolic dimension (LVEDD) and left ventricular end systolic dimension (LVESD) from rest to exercise during knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different levels of submaximal work loads. (mean ± SE)
* $p \leq 0.05$ KE vs. BIC
** $p \leq 0.05$ final repetitions vs. repetitions 5-7

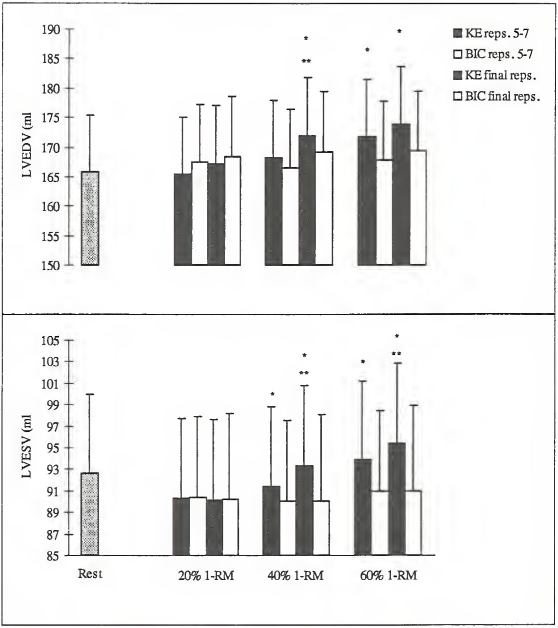


Figure 4-12. Changes in left ventricular end diastolic volume (LVEDV) and left ventricular end systolic volume (LVESV) from rest to exercise during knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different levels of submaximal work loads. (mean ± SE)
* $p \leq 0.05$ KE vs. BIC
** $p \leq 0.05$ final repetitions vs. repetitions 5-7

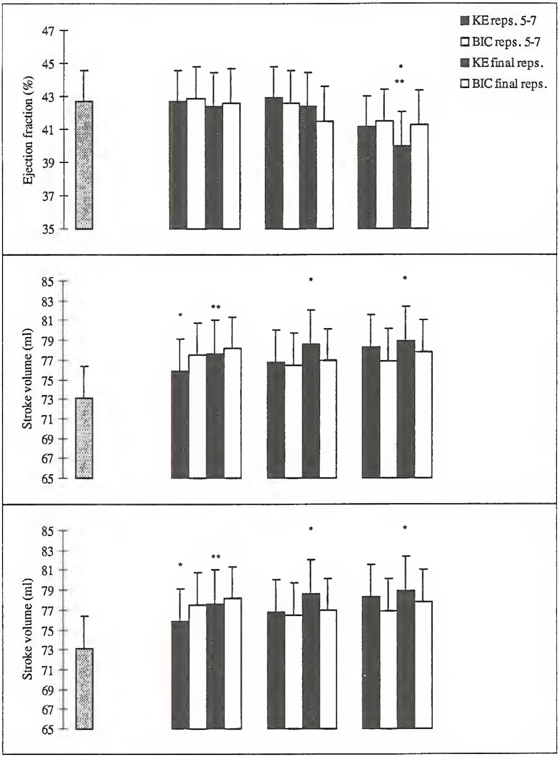


Figure 4-13. Changes in ejection fraction, stroke volume and cardiac output during knee extension (KE) and one-arm biceps curl (BIC) resistance exercise at different levels of work loads. (Mean ± SE).

* p≤0.05 KE vs. BIC

** p≤0.05 final repetitions vs. repetitions 5-7

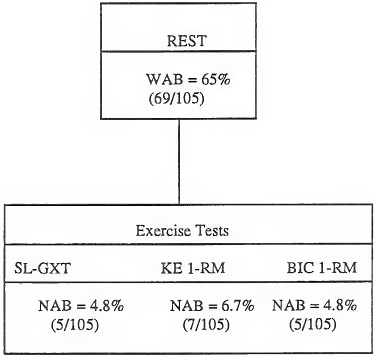


Figure 4-14. Prevalence of resting and exercise-induced wall motion abnormalities.
WAB - Wall Abnormality
SL-GXT - Symptom Limited Graded Exercise Test
KE - Knee Extension
BIC - One-arm Biceps Curl
1-RM - One Repetition Maximum
NAB - New Abnormality

REST

WAB = 65%
(69/105)

	Resistance Exercise					
	KE			BIC		
	20% 1-RM	40% 1 RM	60% 1-RM	20% 1-RM	40% 1-RM	60% 1-RM
Reps. 5-7	No NAB	NAB = 2.8% (3/105)	NAB = 10.5% (11/105)	NAB = 1.9% (2/105)	NAB = 1.9% (2/105)	NAB = 6.7% (7/105)
Final reps.	No NAB	NAB = 4.8% (5/105)	NAB = 12.4% (13/105)	NAB = 1.9% (2/105)	NAB = 2.8% (3/105)	NAB = 10.5% (11/105)

Figure 4-15. Prevalence of resting and exercise-induced wall motion abnormalities at submaximal resistance exercise.
WAB - Wall Abnormality
SL-GXT - Symptom Limited Graded Exercise Test
KE - Knee Extension
BIC - One-arm Biceps Curl
1-RM - One Repetition Maximum
Reps. - Repetitions
NAB - New Abnormality

CHAPTER 5
DISCUSSION

Currently, cardiac patients are being introduced to resistance activities during the first weeks of an outpatients rehabilitation program. Moreover, low-moderate risk patients (e.g. older cardiac patients, patients with moderate LVD and patients with mitral valve prolapse syndrome), who in the past were excluded from resistance training, are now being considered to engage in weightlifting exercise programs. In light of the recent trend towards expanding the cardiac population that can participate in resistance training, the issue of safety of strength testing and resistance exercise becomes a concern. Therefore, the main objectives of this study were to evaluate the safety of strength testing and resistance exercise in low-moderate risk cardiac patients with LVD (30%<EF<49%).

Responses During Strength Testing

A primary purpose of the present study was to establish the safety of strength testing. Maximal strength testing techniques are now recommended for evaluating baseline strength levels, establishing initial weight loads for training and tracking changes in strength over time (AACVPR, 1995; ACSM 1995). Most of the studies previously published on resistance training in cardiac patients used 1-RM tests as the methodology to evaluate muscle strength pre and post resistance training intervention (Butler et al., 1987;

Crozier-Ghilarducci 1989; Daub et al., 1996; Kelemen et al., 1986; McCartney et al., 1991; Stewart et al., 1988; Wilke et al., 1991), or to determine submaximal work loads in order to evaluate cardiovascular responses during resistance exercise (Faigenbaum et al., 1990; Featherstone et al., 1993; Haslam et al., 1988; McKelvie et al., 1995, Stralow et al., 1993; Wiecek et al., 1990). However, few studies have evaluated cardiovascular performance during the 1-RM maneuver (Crozier-Ghilarducci 1989; Daub et al., 1996; Faigenbaum et al., 1990; Featherstone et al., 1993). MacDougall et al. (1985) demonstrated mean BP values of 320 mmHg for SBP and 250 mmHg for DBP during heavy leg press resistance exercise. Thus, due to the abrupt increase in the pressor response with heavy weightlifting (MacDougall et al., 1985; Haslam et al., 1988), it is surprising that so few investigators have measured the hemodynamic responses in cardiac patients while performing 1-RM tests.

Safety of One-Repetition Maximum Test

In agreement with other studies (Daub et al., 1996; Faigenbaum et al., 1990; Featherstone et al., 1993), none of our patients complained of angina, developed myocardial ischemia or demonstrated rhythm abnormalities during and after 1-RM strength tests for both KE and BIC exercise. However, during the SL-GXT six patients (40%) demonstrated ST segment depression that lasted for 5 minutes into recovery. For two of the patients the ischemic changes were coupled with chest pains (2+ on the 4 point Angina Scale). An additional four patients, showed ventricular and atrial ectopies during SL-GXT and into recovery.

We assume that the absence of ischemic changes during the 1-RM test of this study may be the result of lower mean peak RPP values (table 4-3) attained during both 1-RM strength tests as compared to mean RPP values measured during the SL-GXT. Changes in RPP are associated with parallel changes in myocardial oxygen consumption (MVO_2) and coronary BF (Braunwald et al., 1958; Gobel et al., 1977; Nelson et al., 1974). Nelson and associates (1974) examined MVO_2 during dynamic, static and combined static-dynamic exercise in healthy subjects. The authors found that MVO_2 was highly correlated to the products of HR and SBP regardless of whether the BP was obtained by a central aortic catheter ($r = 0.88$) or by indirect BP measurements ($r = 0.85$). In the present study, the low mean peak RPP values were the result of both lower peak HR and SBP values (table 4-3) during 1-RM test, suggesting a lower MVO_2 compared to SL-GXT, probably owing to the limited time for HR and BP to respond during a single movement. However, there is a need to emphasize that the BP values were obtained immediately post exercise, therefore, they do not reflect the real BP values attained during 1-RM maneuver. In addition patients in the current study were taking β-blockers medication that resulted in diminished HR response. Values comparable to those attained in our study were presented by Featherstone et al. (1993) who reported on mean RPP values of 120 $mmHg \cdot min^{-1} \cdot 10^{-2}$ during both BIC and KE resistance exercise compared to 250 $mmHg \cdot min^{-1} \cdot 10^{-2}$ achieved during SL-GXT.

The Valslava maneuver is an integral part of heavy weightlifting, it stabilizes the trunk, and during very heavy lifts, i.e., above 85% of MVC, subjects find it necessary to carry out the maneuver or part of it in order to attain the desired force production.

Previous studies have shown that intrathoracic pressure induced by the Valsalva maneuver immediately relieves myocardial ischemia in cardiac patients (Ewing et al., 1976; Pepine and Wiener, 1979; Pepine and Nichols, 1988). After the initial increase in intrathoracic pressure, LV dimensions remain relatively constant for several beats, while the developed pressure decreases rapidly. With the continued straining the reduced venous return diminishes ventricular size with an additional decrease in the developed pressure. Thus, the increase in intrathoracic pressure results in a decline of some of the hemodynamic determinants (e.g.. pressure and volume) of MVO_2 (Pepine and Nicholas, 1988).

Furthermore, the added intrathoracic pressure due to the Valsalva maneuver is instantly transmitted to the cerebrospinal fluid, so that the cerebrospinal pressure increases to match the pressure in the thorax and abdomen. This may represent a protective mechanism by reducing transmural pressures across cerebral vessels, consequently reducing the risk of vascular damage under the high pressures being elicited from this type of exercise (Lentini et al., 1993; MacDougall et al., 1985; MacDougall et al., 1992). Although the subjects in the present study were instructed to exhale during the initiation of the lift, some degree of increased intrathoracic pressure probably developed as a result of tension development needed for lifting weights at their maximal capacity. However, if indeed such increase in intrathoracic pressure occurred it lasted for very short period of time and did not cause any deleterious effects.

Hemodynamic Responses During Strength Test

It is worth noting that BP measurements during 1-RM tests were taken immediately post exercise. Thus, the genuine BP values reached during the lift were not recorded. Several investigators demonstrated a rapid decline of SBP and DBP immediately at the completion of weightlifting exercise (Haslam et al., 1988; MacDougall et al., 1985; Wiecek et al., 1990). As for the present study, in order to minimize the differences in BP values at the time of measurement compared to the actual values during the lift, the BP cuff was inflated prior to initiation of the test. Faigenbaum et al. (1990) reported significantly lower mean SBP values immediately post resistance exercise (132 ± 14 mmHg) using both lower and upper body exercise, compared to values recorded in the present study for both KE and BIC exercise (table 4-3). The differences between the values can be explained by the different methods in measuring BP. Since both systolic and diastolic BP decline rapidly to resting values within 5-10 seconds post exercise, the inflation time that was saved in the present study may have resulted in higher mean SBP values. Nevertheless, BP measurements were still performed immediately post exercise, therefore, values were considerably low compared to the genuine values attained during the 1-RM maneuver.

An additional procedure used in cardiac rehabilitation settings for muscle strength assessment is the 90% of 1-RM method. This method considers the maximal weight load that can be lifted twice (i.e. 2-RM) as 90% of 1-RM. Using this 90% score, a 1-RM is calculated and used to establish training weights (AACVPR, 1995; Franklin et al., 1991; Kelemen et al., 1989; Sparling et al., 1990). Fleck and Dean (1987) measured intra-arterial BP and cardiovascular responses in young adults during one-arm overhead press and KE resistance exercise at 1-RM and at numerous submaximal loads. The authors demonstrated a significantly greater pressor response during sets carried out to fatigue at various percentages of 1-RM (e.g. 90, 80, 70 and 50%) than that at 1-RM. Therefore, 1-RM tests may impose less of a strain on the heart than was previously presumed, suggesting this method as more favorable compared to other procedures for muscle strength assessment (i.e. 2-RM or 5-RM).

Cardiovascular safety during maximal strength testing performed by 6,653 healthy adults was documented by Gordon and associates (1995). None of the subjects experienced a clinically significant cardiovascular event during the 1-RM test. Accordingly, the findings suggests that the 1-RM strength test is a safe procedure for both KE and BIC exercises in healthy adults and low-moderate risk cardiac patients with LVD.

Left Ventricular Function During Strength Testing

Deterioration in LV function is a more sensitive marker of myocardial ischemia during exercise than is ST segment depression. Echocardiography is a noninvasive reproducible method for estimating LV performance at rest and during exercise. Two-dimensional echocardiography has been found to accurately assess global LV function in patients with regional and diffuse wall motion abnormalities (Albin and Ranko, 1990; Feigenbaum, 1994; Ginzton et al., 1984; Maurer and Nanda, 1981; Robertson et al., 1983).

In accordance with previous data, our patients demonstrated an increase in mean LVEDV during the SL-GXT compared to rest (table 4-5). Comparable changes were observed in both normal subjects (Crawford et al., 1979; Effron, 1989; Keul et al., 1981) and in LVD patients (Donckier et al., 1991; Konstam et al., 1992) demonstrating increased diastolic filling. Previous studies published on LVD patients found no change in mean LVESV values during dynamic exercise compared to rest (Donckier et al., 1991; Konstam et al., 1992; McKelvie et al., 1995). The same was observed in the present study (table 4-5). A Reduction or maintenance of LVESV during dynamic exercise demonstrates that there is a sufficient augmentation in the myocardial inotropic state to overcome the exercise-associated increase in afterload resulting in increased SV and EF (table 4-6) (Crawford et al., 1979; Effron, 1989; Keul et al., 1981; Konstam et al., 1992). Consequently, the increased CO seen in the present study was the result of an increase in both the inotorpic and chronotropic responses.

We are unaware of any published data available on LV function evaluation during 1-RM strength testing in CAD patients. In earlier investigations were hemodynamic and LV function evaluation during static exercise was performed, used MVC solely for determination of submaximal work loads in both healthy and CAD subjects (Crawford et al., 1979; Fisman et al., 1992; Painter and Hanson, 1984; Perez-Gonzales et al., 1981; Sagiv et al., 1985; Seals et al., 1983). Only one review by Keul et al. (1981) on the effect of static and dynamic exercise on LV dimensions, volume and contractility mentioned a decrease in LVEDV during a maximal static contraction. However, the review did not specify on which population the study was performed and which muscle group was involved during the contraction.

In the present study the mean LVEDV values during the KE strength test exhibited a significant increase compared to rest (table 4-5). A comparable response was not observed during the BIC 1-RM test. The difference between the results of the two tests can be explained by the difference in the volume of muscle mass used. The role of the

muscle pump was much more extensive during KE 1-RM resulting in an increased preload. Our patients demonstrated no change in LVESV (table 4-5) during both strength tests suggesting an augmented LV function against an increased pressure load.

The trend for increased (during KE 1-RM) or maintenance (during BIC 1-RM) in mean SV values in our patients coupled with no change in mean EF values (table 4-6) suggests an increase in myocardial contractility. In addition the increase in intrathoracic pressure due to a Valsalva maneuver may assist in cardiac compression resulting in maintenance of SV despite the profound increase in afterload. Therefore, the increase in CO was mediated solely due to an increase in the chronotropic state.

The left ventricular end-systolic pressure-volume relation is used to assess LV contractile performance (Ginzton et al., 1984; Takeuchi et al., 1991). Non invasive evaluation of the LVES pressure/volume ratio was first proposed by Sagawa et al. (1977). The investigators demonstrated that ultrasonic LVD could be an estimate of LVESV and proposed that peak carotid pressure could be an estimate of substituted LVES pressure. Reichek et al. (1982) demonstrated a close correlation between sphygmomanometric SBP and LVES pressure. With the assistance of 2-D echocardiographic analysis for LVESV and sphygmomanometric SBP evaluation during peak dynamic exercise Ginzton et al. (1984) established the accuracy of SBP/LVESV ratio for measuring LV contractility compared to EF in distinguishing between normal subjects and CAD patients. In the present study for both SL-GXT and the KE and BIC 1-RM tests mean SBP/LVESV ratios demonstrated an increase compared to rest, suggesting an increase in LV contractility.

Thus, during the 1-RM maneuver for both upper and lower body exercise the global myocardial function remained stable and no LV deterioration was found.

Responses During Resistance Exercise

Safety of Submaximal Resistance Exercise

The second part of the study was designed to answer the question of how safe for patients with LVD to do low-moderate (20, 40, and 60% using the 1-15 repetition model) resistance exercise? The clinical symptoms manifested in the present study during submaximal resistance exercise were comparable to those previously reported (Crozier-Ghilarducci 1989; Daub et al., 1996; Faigenbaum et al., 1990; Kelemen et al., 1986; Vander et al. 1986). These studies assessed cardiovascular complications that were precipitated during aerobic exercise and found that they occurred as often or more than that were found in resistance exercise. In the present study, only two patients exhibited ischemic changes during resistance exercise compared to six during the SL-GXT. These changes were observed only at the highest intensity (i.e. 60% 1-RM) during KE exercise and were of less magnitude compared to those observed during the SL-GXT. The prevalence of ventricular arrhythmias was not greater during resistance exercise than found during the SL-GXT (five patients vs. four patients, respectively). As previously reported by other investigators, the PVC's and PAC's seen in the present study were found during both the recovery period immediately post exercise (three patients) and the exercise period (two patient) (Daub et al., 1996; Faigenbaum et al,. 1990; Kelemen et al., 1986; Vander et al. 1986).

Attenuation of exercise induced ST depression during combined static-dynamic exercise compared to dynamic exercise alone has been documented by earlier studies. Moreover, mean RPP values at the onset of ST segment changes were statistically higher during static-dynamic exercise than during the dynamic exercise alone (Bertagnoli et al., 1990; DeBusk et al., 1978; DeBusk et al., 1979; Kerber et al., 1975). The hemodynamic responses during weightlifting are considerably different from the responses observed during treadmill exercise. During resistance exercise there is a modest increase in HR response coupled with a substantial rise in SBP, DBP and MAP values. During the SL-GXT, HR and SBP increase substantially with no significant change in DBP values compared to rest. Mean arterial pressure increases relatively little due to an extensive metabolic drive causing vasodilatation (Bezucha et al., 1982; Lewis et al., 1983; Lewis et al., 1985). In the present study hemodynamic responses demonstrated a comparable pattern as found in other studies using resistance exercise (tables 4-3, 4-8, and 4-10). Previous studies have suggested that the increase in diastolic BP may serve as a protective mechanism through an increase in subendocardium perfusion (DeBusk et al., 1979; Kerber et al., 1975; Lowe et al., 1975).

In an *in situ* heart preparation Braunwald et al. (1958) induced a 196% increase in coronary BF and a 164% increase in MVO_2 by increasing heart work through elevating aortic pressure. However, increasing the heart work through augmenting CO resulted in elevation of only 35% in coronary flow and 62% increase in MVO_2. Furthermore, during the course of the CO elevation the DBP decreased, but a rise in diastolic BP was found when the aortic pressure was increased. Nelson et al. (1974) demonstrated higher systolic and DBP, MVO_2 and coronary BF with an addition of static load to dynamic exercise compared to dynamic exercise alone. This suggests an improved myocardial perfusion pressure during exertion associated with an increased pressure load.

In accord with previous studies, mean RPP values during the present study were significantly lower during submaximal resistance exercise compared to treadmill exercise (figure 4-10), suggesting a lower MVO_2 (Faigenbaum et al., 1990; Featherstone et al. 1993; Haslam et al. 1988). The lower mean RPP data were mainly the result of a moderate elevation in HR response during resistance exercise. Barnard et al. (1973) emphasized the importance of HR and DBP duration (diastolic pressure-time index) as a major determinant of myocardial BF and ischemia during sudden exercise in healthy adults. The diastolic pressure-time index is the product of DBP, HR and diastolic time interval. The diastolic time interval can be determined from an ECG tracing by measuring the distance from end of the T wave to the onset of the next QRS complex (Buckberg et al., 1972). Featherstone and associates (1993) demonstrated a higher diastolic pressure-time index with resistance exercise compared to treadmill exercise. The authors used the diastolic pressure-time index to RPP ratio as an indirect estimate of the balance between myocardial oxygen supply-demand. The mean diastolic pressure-time index to RPP ratio values were significantly higher during resistance exercise than found during aerobic exercise. Consequently, improved myocardial oxygen balance during weightlifting may result from a longer diastolic filling time due to a lower HR coupled with a higher DBP.

Hemodynamic Responses During Resistance Exercise

The increased hemodynamic responses seen during weightlifting exercise are the result of a mechanical compression of the blood vessels with each muscle contraction coupled with a pressor response. The pressor response is associated primarily with an elevated SBP, DBP, HR and CO, and to a lesser extent to vasoconstriction in the non-exercising vascular beds (Bezucha et al., 1982; Ewing et al., 1976; Helfant et al., 1971; MacDougall et al., 1985; Misner et al., 1990). The cardiovascular response to resistive exercise is regulated by both central and peripheral mechanisms. The central drive is the trigger for the increased pressor response seen at the initiation of exercise and is also involved in determining the magnitude of the cardiovascular response achieved during exercise. The peripheral mechanism involves activation of nerve endings in the contracting muscles, which in turn activate the medullary cardiovascular center. The rise in BP and HR depend on the duration, relative intensity (percent of MVC), and the total of active muscle mass involved in the exercise (Blomqvist et al., 1981; Lewis et al., 1983; Mitchell et al., 1980; Perez-Gonzalez, 1981; Seals et al., 1983; Tesch et al., 1988).

Mean BP values attained in the present study during KE and BIC submaximal resistance exercise were relatively high compared to those observed in previous studies (tables 4-8 and 4-10). Sparling and colleagues (1990) found no significant differences in mean SBP and DBP values immediately post resistance exercise compared to rest. Accordingly, Crozier-Ghilarduci et al. (1989) reported no change in mean BP values when performing heavy resistance exercise training (80% 1-RM). Kelemen et al.(1986) showed mean values of only 141±20 mmHg during circuit weight training at 40% of 1-RM.

However, these measurements of BP most likely did not reflect the true values generated during the set, since BP data were collected immediately post exercise. Studies using direct BP evaluation demonstrated a rapid decrease in SBP and DBP toward resting values within 5-15 seconds immediately post resistance exercise (Haslam et al., 1988; MacDougall et al., 1985; Wiecek et al., 1990), thus, interpretations of these data should be made with caution.

Wiecek et al. (1990) compared direct and indirect measures of arterial pressure during weightlifting in CAD patients. Indirect mean SBP values both at rest and during leg press exercise was 13% less than the mean SBP recorded directly. Mean indirect SBP recorded immediately post exercise was 31% lower than values attained directly during the actual lift. Mean DBP at rest and during lifting was similar using either method. During the present study even though an indirect BP evaluation technique was used, a tendency comparable to that of the direct method was observed (tables 4-8 and 4-10).

Blood pressure measurements were performed twice during each set - at the midpoint of the set and during the final repetitions of the set - and immediately post exercise. There was a significant reduction in mean SBP and DBP values obtained immediately post exercise compared to the end of the exercise bout for both KE and BIC resistance exercises (figures 4-4 and 4-6, respectively). Thus, for BP response follow-up of cardiac patients during a resistive training session, it is necessary to evaluate BP during the actual exercise bout and not immediately post exercise.

The rapid drop in BP immediately post resistance exercise is most likely caused by the sudden perfusion of vasodilated muscle mass, which was previously occluded, and as a result of a transient pressure undershoot initiated by baroreceptor and cardiopulmonary reflexes responding to the profound increase in BP during exercise (MacDougall et al. 1985). This might cause a sensation of faintness or dizziness, thus, cardiac rehabilitation personnel should instruct patients to recover for several seconds before continuing to their next prescribed exercise. However, our patients did not experience this sensation due to the lower work loads used in the current study compared to MacDougall et al. (20, 40 and 60% 1-RM vs. 90% to fatigue, respectively).

For both KE and BIC resistance exercise the HR and BP responses showed significant increases throughout the set across all submaximal work loads (tables 4-8 and 4-10, respectively). This response was in agreement with previous findings observed in healthy and CAD subjects (Fleck and Dean, 1987; MacDougall et al., 1985; Wiecek et al., 1990). MacDougall et al. (1985) performed direct BP measurements in young healthy adults performing both upper and lower body heavy weightlifting exercises. The cardiovascular responses demonstrated a progressive increase in mean HR, SBP and DBP values with each subsequent repetition. Fleck and Dean (1987) found that mean peak HR was achieved during the last two to three repetitions of the set. Wiecek et al. (1990) also reported that the highest intra-arterial pressures were found during the final repetitions of the set.

As the subject performs more repetitions and begins to fatigue, there is additional motor unit recruitment and increasing involvement of accessory muscles. This results in increasing active muscle mass generating a mechanical compression on the vascular bed in the exercising limbs, consequently leading to an increased BP. In addition to the increased

BP values seen over time, there is a corresponding increase in CO mainly due to the increase in HR throughout the set since changes in SV are very small (tables 4-14 and 4-15) (Bezucha, et al., 1982; Lewis et al., 1985; MacDougall et al., 1992).

Our data demonstrated significant differences in the hemodynamic responses in KE vs. BIC resistance exercise. Submaximal KE resistance exercise resulted in higher hemodynamic values compared to those attained during submaximal BIC resistance exercise (figures 4-7 through 4-9). Previous studies have demonstrated that the magnitude of BP and HR responses were related to the muscle mass involved during static and dynamic resistance exercise (Lewis et al., 1985; MacDougall et al., 1985; MacDougall et al., 1992; Misner et al., 1990; Seals et al., 1983). Seals et al. (1983) demonstrated a positive relationship between the magnitude of the increase in BP and HR response and the size of active muscle mass at the same relative percentage of MVC.

As previously described the cardiovascular responses to resistance exercise are the product of the mechanical compression and the pressor response. Since contraction of a larger muscle mass exerts compression on a greater portion of vasculature, therefore, the BP elevation produced entirely by mechanical compression is proportional to the active muscle mass. In addition, the central and peripheral mechanisms are linked to increased BP and HR and elicits a greater response with a larger muscle mass involvement. The greater the contracting muscle size, the greater is the number of motor units being activated by the central command. The large contracting muscle mass also elicits greater peripheral input from skeletal muscle nerve endings (MacDougall et al. 1985; Mitchell et al. 1981; Seals et al. 1983).

Previous studies (Haslam et al. 1988; Featherstone et al. 1993; Stralow et al., 1993) compared the hemodynamic responses during different submaximal workloads in CAD patients. Our data, using three different work loads, demonstrated a significant increase in mean peak values for HR, SBP and DBP measured during BIC submaximal resistance exercise at 20, 40 and 60% of 1-RM (table 4-10). These data are in agreement with the Haslam et al. (1988) investigation. Using the direct BP measurement technique Haslam et al. demonstrated a significant increase in mean SBP and DBP values for single- and double-leg press and BIC resistance exercises across 20, 40 60 and 80% of 1-RM. However, our hemodynamic data for KE submaximal resistance exercise (table 4-8) are inconsistent with Haslam et al. (1988), which demonstrated no significant difference in mean peak SBP values for 40 and 60% of 1-RM (173 and 177 mmHg, respectively).

On the other hand, our KE resistance exercise data were in agreement with the Featherstone et al. (1994) study. The investigators using the indirect BP measurement technique, did not demonstrate a consistent increase in hemodynamic values for KE resistance exercise from 60% to 80% 1-RM. Since during weightlifting the magnitude of BP response is related to the intensity of the effort, thus, an increase in BP values would be expected with the increase in work load for each resistance exercise bout. It can be speculated that the different measuring techniques used in during the studies, i.e. the direct method in Haslam et al. (1988) vs. the indirect BP measurement in the present study produced the discrepancy between the results.

Our mean TPR values for both resistance exercises and treadmill exercise (tables 4-3, 4-9 and 4-11) correspond with the mean TPR data obtained from healthy subjects

(Bezucha et al., 1982; Lewis et al., 1983; Lewis et al., 1985). During dynamic exercise TPR decreases compared to rest due to local metabolic mediated vasodilatation. However, during resistance exercise mean TPR does not differ from resting values, since the metabolically vasodilatation has a minor impact on systemic resistance when the active muscle mass is small (Bezucha et al., 1982; Lewis et al., 1983; Lewis et al., 1985). In the present study, the mean TPR values immediately post exercise showed a significant reduction in TPR compared to rest at each submaximal exercise bout exhibited. This reduction was mediated by the sudden perfusion of the vasodilated muscle mass, which was previously occluded during the set. This might explain the lower mean TPR values seen during the KE and BIC 1-RM test compared to rest (table 4-3). Due to the fact that the BP values were obtained immediately post exercise, thus, withdrawal of the mechanic vessels compression and reperfusion of vasodilated muscle mass resulted in reduction in systemic resistance.

Mean MAP values for both KE and BIC resistance exercise (table 4-9 and 4-11, respectively) also increased with the increase in relative intensity and with the increase in muscle mass (figure 4-8). These changes in mean MAP are comparable to data obtained in previous studies in both healthy and CAD patients (Bezucha et al., 1982; Haslam et al., 1988; Lewis et al. 1985; Miles et al., 1987; Pianter and Hanson, 1984). During dynamic exercise which involves a large muscle mass, the metabolic vasodilatation is extensive resulting in increased systemic conductance. In contrast, in small muscle mass exercise local vasodilatation has a small effect on systemic conductance resulting in pronounced increase in MAP. Mean arterial pressure response is proportional to the product of CO and TPR. Consequently it can be concluded that the increase in mean MAP values seen in our patients resulted mainly from the increase in CO and not by an increase in total peripheral resistance (tables 4-9, 4-11, 4-14, 4-15).

Left Ventricular Function During Resistance Exercise

Documentation of LV function during resistance exercise in the LVD patients is limited (Chang et al., 1994; McKelvie et al., 1995). However, during the 70s and early 80s a large number of investigators performed assessments of LV performance during static exercise in normal and CAD patients (Crawford et al., 1979; Ewing et al., 1976; Kivowitz et al., 1971, Perez-Gonzales et al., 1981; Sagiv et al., 1985; Seals et al., 1983)

During KE resistance exercise our patients exhibited an increase in mean LVEDV values during the final repetitions of both 40 and 60% of 1-RM (table 4-12). These findings are consistent with the previous studies performed in LVD patients during dynamic exercise (Donckier et al., 1991; Konstam et al., 1992). Weightlifting consists of both dynamic (overcoming the inertia of the weight along the full range of motion) and static components (Lentini et al., 1993; MacDougall et al., 1985). Therefore, the hemodynamic responses to resistance exercise corresponds to both types of exercise.

None of the previous studies assessed LV function during upper body exercise. Since most of daily living activities involve upper body exertion such as pushing, pulling and carrying, it is important to obtain information during upper body resistance exercise. Mean LVEDV values during BIC resistance exercise were significantly lower compared to KE resistance exercise (table 4-13, figure 4-12). Since BIC exercise involved a single small muscle group, it placed a lower demand on cardiovascular system compared to the

KE exercise. Moreover, during the KE exercise, the muscle's contraction and relaxation resulted in an increased venous return due to a larger muscle pump compared to the BIC resistance exercise.

In the present study changes in mean LVESV values during KE submaximal work loads exhibited a pattern resembling a submaximal static exercise (table 4-12). Mean LVESV decreased significantly during light resistance exercise (20% 1-RM). As the work load increased to 40 and 60% 1-RM there was an increase in mean LVESV values. Previous studies using a submaximal static exercise in healthy subjects demonstrated comparable findings. Keul et al. (1981) found a decrease in LVESV during light static exercise (handgrip (HG) performed at 30% of MVC). While, Crawford et al. (1979) found an increase in LVESV in healthy subjects performing HG at 50% of MVC to fatigue. The increase in mean LVESV values can be explained on the account of increased afterload, which is related to the increase in exercise intensity and muscle mass involved. In the present study mean LVESV values during BIC resistance exercise did not alter throughout the set and among the sets (table 4-13, figure 4-12). It seems that the magnitude of hemodynamic strain opposing the heart during BIC resistance exercise did not show the same response as found in static exercise.

McKelvie et al. (1995) did not find changes in mean LVEDV and LVESV values in LVD patients performing a single leg press at 70% of 1-RM. The differences between the studies can be attributed to differences in the patient population. In the current study the patients were classified as class I and class II by the New York Heart Association, i.e. patients without symptoms at rest and with or without symptoms during ordinary activity.

Whereas, in McKelvie et al. study, the patients were classified as class II and class III with a higher degree of LVD compare to our patients (mean EF values were 27 and 42%, respectively). The same variance in LV function was documented by other studies demonstrating enhanced cardiac reserve during exercise in patients with mild LVD (Chang et al., 1994; Elkayam et al. 1985; Kivowitz et al., 1971; Konstam et al. 1992).

In the present study, a small increases in mean SV were observed for both KE and BIC resistance exercise across all intensities (tables 4-14 and 4-15). These results are in agreement with previous studies of patients with LVD performing static and resistance exercise (Chang et al., 1994; Kivowitz et al. 1971). Both studies divided thier patients into two categories; patients with mild and severe disease. Chang and associates (1994) demonstrated an increase in SV in patients with mild LVD during KE resistance exercise at 70% of 1-RM whereas patients with severe LVD demonstrated a reduction in SV. Kivowitz et al. (1971) found that while performing HG exercise at 25% of MVC for 5 minutes, patients classified as class I and class II by the New-York Heart Association demonstrated an increase in LV stroke work index with a small or no increase in LV end diastolic pressure.

The utility of using EF as a measure of ventricular function has been questioned since it is dependent on both preload and afterload. In contrast, the LV pressure/volume relation at the end of systole has been demonstrated to have the advantage of being minimally influenced by preload and afterload over a wide range of preload and afterload conditions (Ginzton et al., 1984; Grossman, et al., 1977; Sagawa et al., 1977). Noninvasive determination of the LV volume/pressure relation by using SBP and LVESV was found to accurately assess LV function (Ginzton et al., 1984; Grossman, et al., 1977; Reichek et al., 1982; Sagawa et al., 1977). This might explain the discrepancy seen in the present study between mean EF values and mean SBP/LVESV ratios (table 4-14). There was a small but significant reduction in mean EF values during the last set (60% 1-RM) in KE resistance exercise suggesting a reduction in LV function. Whereas, mean EF values during BIC resistance exercise did not demonstrate significant changes between and within work oads (table 4-15, figure 4-13). In contrast to this observation our patient's mean SBP/LVESV ratio demonstrated a significant increase throughout the whole set (tables 4-14 and 4-15). Theses findings are in agreement with Mckelvie et al. (1995) who found an increase in mean SBP/LVESV with no change in mean LVEDV, LVESV and EF values during one leg-press at 70% 1-RM in LVD patients, suggesting that resistance exercise did not adversely affect LV function.

In accordance with previous studies performed in both healthy subjects (Lentini et al.; 1993; MacDougall et al., 1985; Miles et al.,1987) and CAD patients (Mckelvie et al. 1995; Sagiv et al. 1985) the mean CO values increased significantly during both modes of resistance exercise (tables 4-14 and 4-15). The increase was primarily mediated by an increase in HR since the increase in SV was relatively small. Corresponding with to the changes in HR during the exercise bouts differences in CO (figure 4-13) were found between KE and BIC exercise and among intensities.

Exercise-Induced Wall Motion Abnormalities

To the best of our knowledge, none of the previous studies performed echocardiographic wall motion assessment during 1-RM strength testing and resistance exercise. Butler et al., (1987) evaluated segmental wall motion in low risk cardiac patients immediately post circuit weight training. Fisman and colleagues (1992) used isometric exercise (50% of MVC exerted on 175-pound Bullworker telescopic dynamometer bar) for assessing LV wall motion patterns.

One of the advantages of 2-D echocardiography is that the technique provides a noninvasive method for assessing regional LV function. This technique is able to visualize portions of the LV that are hidden from view by means of contrast arteriography (Agati et al., 1991; Feigenbaum, 1994; Maurer et al., 1981; Sheilkh et al., 1990).

In the present study, prevalence of new wall motion abnormalities during both KE and BIC 1-RM strength testing were comparable to those observed during SL-GXT (4.8, 6.7 and 4.8% increase in new wall motion abnormality, respectively) (figure 4-14). During submaximal resistance exercise a moderate increase in new wall motion abnormalities was observed with the increase in exercise intensity (figure 4-15). Similar patterns were observed for both KE and BIC exercises. However, the occurrence of new wall motion abnormalities was slightly higher during KE resistance exercise compared to BIC (12.4 and 10.5% increase in new wall motion abnormality during 60% 1-RM, respectively). This can be explained by the higher active muscle mass involved during KE exercise compared to BIC resulting in greater hemodynamic responses. Our data are in agreement with Sagiv et al. (1985) who used the radionuclide angiography technique and demonstrated an increase in new wall motion abnormalities during isometric exercise with an increase in active muscle mass in CAD patients. During 30% MVC of HG exercise new wall motion abnormalities developed in two patients out of 14, whereas during the deadlift exercise new wall motion abnormalities occurred in 13 patients.

Fisman et al. (1992) compared isometric exercise (50% of MVC) with SL-GXT performed on a cycle ergometer in eliciting LV wall motion abnormalities in low risk cardiac patients post myocardial infarction (EF ≥ 55%). The authors found that the isometric exercise-induced wall motion abnormalities were of a severity proportional to the degree of coronary narrowing. Moreover, the isometric exercise modality was comparable to dynamic exercise in identifying obstructions in a specific vessel.

Contrast findings were demonstrated by Butler et al. (1987). The investigators found in CAD patients a worsening of wall motion in 5 out of 61 segments during aerobic training (85% of maximal HR) compared to only one segment immediately post circuit weight training at 40-60% of 1-RM. Echocardiographic evaluations were begun within 1 minute post exercise and were completed within 5 minutes. The contrast findings between the present study compared to those observed in Butler et al. investigation may be due to the different time frame of data collection (during resistance exercise vs. immediate post exercise) and differences in the patient populations (LVD patients vs. low risk patients, respectively).

Summary

Since the beginning of the 90s there has been an increasing awareness of the favorable effects of resistance training as a tool for promoting public health. Studies have shown health benefits associated with the use of resistance training for both the healthy and diseased populations. Furthermore, data have demonstrated the safety and efficacy of resistance training in low risk cardiac patients. Consequently, the recent guidelines of the AHA, ACSM and AACVPR published in 1995 included specific guidelines for strength training prescription for low risk cardiac patients.

In the present study, the safety of strength testing and resistance exercise was evaluated for both upper and lower body exercise in low-moderate risk cardiac patients. Our patients demonstrated no angina or electrocardiographic ischemic changes during 1-RM strength testing compared to the SL-GXT. These absences in ischemic signs and symptoms were due to significantly lower mean peak RPP values attained during both 1-RM strength tests as compared to mean RPP values measured during the SL-GXT, suggesting a lower MVO_2. The lower RPP values seen in the present study were the result of lower mean values of both HR and SBP attained during 1-RM tests compared to the SL-GXT. However, there is a need to emphasize that the BP values were obtained immediately post exercise, therefore, they do not reflect the real BP values attained during 1-RM maneuver. In addition patients in the current study were taking β-blockers medication that resulted in diminished HR response.

Echocardiographic evaluation of LV function during 1-RM strength testing demonstrated a maintenance of global LV function. The increased percentage in new wall motion abnormalities were similar across all exercise tests, suggesting that the 1-RM test did not cause higher cardiac stress when compared to the SL-GXT, which is contrary to what has been previously presumed.

Resistance exercise performed at different submaximal work loads (20, 40 and 60% of 1-RM) appeared to be safe in our specific patient group. Hemodynamic responses (i.e. HR, BP and RPP) were within the range of 60 to 85% of their peak values attained during SL-GXT. These values are in the range that is recommended for aerobic exercise prescription for cardiac patients. Moreover, the increase in BP seen in our patients appeared to be largely the result of increased CO and not due to an increase in peripheral resistance.

Left ventricular function demonstrated a slight increase during both resistance exercises by echocardiographic means. There was a small but significant decrease in EF values during the final set (60% 1-RM) of KE exercise compared to rest (40 vs. 42%, respectively). In addition a moderate increase in the occurrence of wall motion abnormalities was observed during the highest submaximal work load for both KE and BIC resistance exercise. Nevertheless, there were no adverse effects on LV function. Values of SBP/LVESV ratio were 2.1 during KE 60% 1-RM resistance exercise vs. 1.5 during rest suggesting enhanced LV contractility.

Most every day activities such as carrying, pulling and pushing are performed by the upper extremities, therefore an increase in upper body muscle strength and endurance is important and should aid the patients in performing these tasks using a reduced percentage of their 1-RM force. Thus, reducing the risk for the precipitation of a cardiac event or a musculoskeletal injury. The present study demonstrated no adverse effect on cardiovascular performance during arm exercise compared to leg exercise. Moreover, hemodynamic responses were significantly lower during BIC resistance exercise compared to KE suggesting a lesser likelihood for cardiac complications.

There are several limitations to the study. Continuous, intraarterial pressure measurement is the most accurate and reliable method for measuring BP. However, due to inherent risk of arterial catheterization in LVD patients, indirect measurements of BP were utilized. Echocardiographic analyses were performed by only one observer, since qualitative assessment is subjective, thus, two to three observers are recommended. The relatively small number of patients participating in the study, the absence of women and the fact that only two different resistance exercises were used needs to be considered.

Conclusions

The purpose of the study was twofold: 1) to establish the safety of 1-RM strength testing, and 2) to establish the safety of repetitive resistance exercise performed at various submaximal intensities using 10-15 repetitions at 20, 40 and 60% of 1-RM in low-moderate risk cardiac patients with LVD (30% < EF < 49%).

The data of the present study support the first hypothesis that 1-RM strength testing is safe and does not impose any adverse effects upon cardiac function in low-moderate risk cardiac patients.

Low-moderate risk cardiac patients can safely engage in resistance training using 10-15 repetitions at 20, 40 and 60% of 1-RM. Notwithstanding, a small increase in occurrence of ischemic changes, ventricular arrhythmias, EF reduction and higher prevalence of new wall motion abnormalities during the highest exercise work load (i.e.

60% of 1-RM) and with larger muscle mass involved (KE vs. BIC). However, theses findings are small in magnitude and do not suggests reduced cardiac performance. Thus, the second hypothesis can be accepted. The present study demonstrated no adverse effect on cardiovascular performance during arm exercise compared to leg exercise. Moreover, hemodynamic responses were significantly lower during BIC resistance exercise compared to KE suggesting a lesser likelihood for cardiac complications, hence, supporting the study's third hypothesis.

Implication For Future Research

1. The present study was performed on a small sample size, thus, it is recommended to perform the current protocol with a larger sample and with additional resistance exercises.
2. The indirect BP evaluation technique performed in the present study resulted in lower BP values compared to the true values attained during 1-RM tests and resistance exercise. A study with direct BP measurements will provide additional information regarding hemodynamic responses during strength testing and resistance exercise.
3. Further research is suggested using female cardiac patients with LVD.
4. The information available on hemodynamic responses and LV function is limited to young male adults and low risk cardiac patients, therefore there is a need for a comparable study performed on healthy aged matched male and female subjects.
5. The patient population used in the present study demonstrated moderate LVD, i.e. the average EF was 42% (EF ranged from 30 to 49%). It is recommended to assess LV

function responses to 1-RM strength testing and resistance exercise in patients with more reduced LV function.

6. Fleck et al. (1987) demonstrated reduced pressor responses in healthy young adults who engaged regularly in resistance exercise training compared to novice participants. Therefore, future research is needed to evaluate the hemodynamic responses in patients with LVD to resistance training at different submaximal work loads.

APPENDIX A
24-HOUR HISTORY
(ENGLISH AND HEBREW VERSIONS)

24-HOUR HISTORY

NAME__________________________ DATE______________ TIME______________

How much sleep did you get last night? (Please circle one)

1 2 3 4 5 6 7 8 9 10 (Hours)

How much sleep do you normally get? (Please circle one)

1 2 3 4 5 6 7 8 9 10 (Hours)

How long has it been since your last meal or snack? (Please circle one)

1 2 3 4 5 6 7 8 9 10 11 12 13 14 (Hours)

List the meals or snack eaten:

____

When did you last have:

A cup of coffee or tea ____________________________

Smoke a cigarette, cigar, or pipe ____________________________

Drugs (including aspirin) ____________________________

Alcohol ____________________________

Last time donated blood ____________________________

Any recent illness ____________________________

Suffer from respiratory problem ____________________________

What sort of physical exercise did you perform yesterday?_______

____

What sort of physical exercise have you performed today?_______

____

Describe your general feelings by checking one of the following:

__________ Excellent

__________ Very good

__________ Good

__________ Neither bad or good

__________ Bad

__________ Very bad

__________ Very, very bad

__________ Terrible

24 שעות אחרונות

שם הנבדק - ______________________

תאריך - ______________ שעה - ___________

כמה שעות ישנת בליל אמש ? (אנא הקף בעיגול מספר אחד בלבד)

1 2 3 4 5 6 7 8 9 10 (שעות)

כמה שעות שינה ללילה הנך בדרך כלל ישן ? (אנא הקף בעיגול מספר אחד בלבד)

1 2 3 4 5 6 7 8 9 10 (שעות)

כמה זמן חלף מאז שאכלת לאחרונה (אנא הקף בעיגול מספר אחד בלבד)

1 2 3 4 5 6 7 8 9 10 (שעות)

נא ציין את מה שאכלת:

____

מתי לאחרונה:

שתית כוס קפה או תה ______________________

עישנת סיגריה, סיגר או מקטרת ______________________

לקחת תרופות ______________________

שתית אלכוהול ______________________

מתי בפעם האחרונה תרמת דם ______________________

מתי חלית לאחרונה ______________________

מתי לאחרונה סבלת מבעיות נשימה ______________________

איזו פעילות גופנית ביצעת אתמול ? ______________________

____

איזו פעילות גופנית ביצעת היום ? ______________________

____

אנא תאר את הרגשתך הכללית על ידי סימון אחד

מצוין _____

מאד טוב _____

טוב _____

לא טוב ולא רע _____

רע _____

רע מאד _____

מאד מאד רע _____

איום _____

APPENDIX B
INFORMED CONSENT FORM
(ENGLISH AND HEBREW VERSVIONS)

IRB# 545-94

Informed Consent to Participate in Research

J. Hillis Miller Health Center
University of Florida
Gainesville, Florida 32610

You are being invited to participate in a research study. This form is designed to provide you with information about this study. The principal investigator or representative will describe this study to you and answer any of your questions. If you have any questions or complaints about the informed consent process or the research study, please contact the Institutional Review Board (IRB), the committee that protects human subjects, at (352) 392-1494.

1. **Name of Subject**

2. **Title of Research Study**
   Acute Hemodynamic Responses to Strength Testing and Resistance Exercise in Patients with Left Ventricular Dysfunction.

3. a. **Principal Investigator(S) and Telephone Number(S)**

   Michael L. Pollock, Ph.D. (352) 392-9575

   Co-Investigator: William F. Brechue, Ph.D.
   Sagiv Michael, Ph.D
   Ehud Goldhammer, M.D.
   Galila Werber, M.S.
   Anat Shaar
   David T. Lowenthal, M.D., Ph.D.

Two. Sponsor of the Study (if any)

None.

4. The Purpose of the Research

The purpose of this research is to contribute information concerning resistance testing and resistance training that can be used to help exercise prescription in heart patients who have moderate damage to the heart.

5. Procedures for This Research

This study will last 5 weeks with a minimum of five visits (about 2 hours each visit). Testing will be performed at the Cardiac Rehabilitation Program at the Wingate Institute, Israel. Visit 1 will be an orientation. You will receive a full explanation of the study, including its benefits and risks. You will also be asked to read and if willing sign this informed consent form. During this visit you will also undergo a medical evaluation and screening, and body fat measurements. Resting and exercise cardiac function, during a diagnostic treadmill graded exercise test, will be measured by electrocardiography (ECG) and echocardiography (a picture of your heart without radiation). Your heart beat and blood pressure will be monitored continuously during the exercise test. A cardiologist will be present for this test. This visit will allow us to assess your physical health status and see if you meet the criteria for entry into the study.

On visit 2, a maximal treadmill graded exercise test will be performed. This test involves walking on a treadmill until you are maximally fatigued. This test will last about 10 minutes. During this test, your heart rate, blood pressure, and breathing will be monitored. You will wear headgear with an attached mouthpiece to monitor your breathing. This apparatus will collect your expired air, which will be used to determine the maximum amount of oxygen that you can use.

During visit 3 you will perform a strength tests. The test will include lifting a maximum weight that you can lift one times (1-RM). This visit will include a 1-RM test on an upper body (one-arm curl with free weights) and a lower body machine (leg extension weight machine). During this test, your heart, blood pressure, and cardiac function will be monitored. The test will begin with an active warm-up, lifting a light weight 6 to 8 times. You will start with a light weight, and 5 to 10 pounds will be added to each lift. Exactly how much weight is added will depend on how easy the previous lift felt. You will rest between each attempt until you have returned to your resting heart rate and blood pressure values. It usually takes 4-5 attempts to complete a 1-RM test. Your heart rate will be monitored by ECG and cardiac function will be monitored by echocardiography. If any abnormalities occur the test will be stopped. Also during visit 3 you will perform one bout of one-arm and leg exercise at a low work load of 20% of your maximal lift effort for 15 repetitions. During the test, your heart rate, blood pressure, and cardiac function will be monitored.

During visit 4 you will perform several bouts of upper body and lower body resistance exercise at various weight loads (40%, and 60% of maximal effort). You will perform 10-12 repetitions at each intensity. There will be about a 5 minutes rest between each exercise intensity. The weight loads lifted will be based on your 1-RM strength test results. During these tests, your heart rate, blood pressure, and cardiac function will be monitored.

Visit 5 will be a repeat of the visit 4 procedures and will be exactly the same.

6. Potential Risks or Discomforts

Treadmill graded exercise testing is associated with a small risk of heart attack. The overall cardiac complications rate in patients is approximately 4 complications per 10,000 tests, and only 1 death per 20,000 tests. The risk stated refers to overall and general populations and that this risk might be higher in this group of individuals. The risks will be minimized in this study through extensive health screening done prior to testing. Also, a physician will be present for the entire test. Due to the potential risk of an unexpected cardiac emergency, a "Crash Cart" with all appropriate medications and defibrillator will be on hand in the graded exercise test room. Subjects may expect fatigue, breathlessness, and muscle soreness accompanying the exercise testing. This is normal and temporary.

Recently reviewed studies present evidence that resistance training appears to be safe in coronary artery disease patients with heart problems that don't require hospitalization, and left ventricular function that works well enough . Most research reports noted minimal increases in heart rate and arterial blood pressure. It is common to experience muscle soreness one to two days after resistance testing and training. This is temporary and normal and will not interfere with normal daily activities. Exercise difficulty will progress from low to moderate weights, and it will be monitored for safety.

If you wish to discuss these or any other discomforts you may experience, you may call the Principal Investigator listed in #3 of this form.

7. Potential Health Benefits to You or to Others

The benefits for your participation in this study include a physical evaluation performed by a physician, including an evaluation of your heart during exercise to exhaustion or muscle failure on a treadmill, resistance exercise, and determination of upper and lower body fat, and skeletal muscle strength. We hope that the data from this study will help in the establishment of guidelines for resistance exercise appropriate for heart patients like yourself, with weak heart muscle. We will also provide you with instructions for starting or continuing and exercise program.

8. Potential Financial Risks

There are no financial risks associated with your participating in this study.

9. Potential Financial Benefits to You or to Others

There are no financial benefits associated with your participating in this study.

10. Compensation for Research Related Injury

In the unlikely event of you sustaining an physical or psychological injury which is proximately caused by this study:

_X_professional medical; or____professional dental; or____professional consultative

care received at the J. Hillis Miller Health Center will be provided without charge. **However, hospital expenses will have to be paid by you or your insurance provider.** You will not have to pay hospital expenses if your are being treated at the Veterans Administration Medical Center (VAMC) and sustain a physical injury during participation in VAMC-approved studies.

11. Conflict of Interest

There is no conflict of interest involved with this study beyond the professional benefit from academic publication or presentation of the results.

12. ALTERNATIVES TO PARTICIPATING IN THIS RESEARCH

You are free not to participate in this study. If you choose to participate, you are free to withdraw your consent and discontinue participation in this research study at any time without this decision affecting your medical care. If you have any question regarding your rights as a subject, you may phone the Institutional Review Board (IRB) office at (352) 392-1494

13. Withdrawal From This Study

If you wish to stop your participation in this research study for any reason, you should contact Michael L. Pollock, Ph.D. at (352) 392-9575. You may also contact the Institutional Review Board (IRB) office at (352) 392-4646

14. Confidentiality

The University of Florida and the Veterans Administration Medical Center will protect the confidentiality of your records to the extent provided by Law. You understand that the Study Sponsor, Food and Drug Administration or the Institutional Review Board have the legal right to review your records.

15. ASSENT PROCEDURE (if applicable): [Assent is the procidure to obtain agreement to particiapte in the research from a subject, such as a child, who cannot five local consent]

Not applicable

16. Signatures

____

Subject's Name

The Principal or Co-Principal Investigator or representative has explained the nature and purpose of the above-described procedure and the benefits and risks that are involved in the research protocol.

__________________________________ __________

Signature of Principal or Co-Principal Investigator or representative obtaining consent — Date

You have been fully informed of the above-described procedure with its possible benefits and risks and you have received a copy of this description. You have given permission for your participation in this study.

__________________________________ __________

Signature of Subject or Representative — Date

If you are representing the patient or subject, please print your name ______

________________________ and indicate one of the following:

- ____ The subject's parent
- ____ The subject's guardian
- ____ A surrogate
- ____ A durable power of attorney
- ____ A proxy
- ____ Other, please explain:

__________________________________ __________

Signature of Witness — Date

If a representative signs and if appropriate, the subject of this research should indicate assent by signing below.

__________________________________ __________

Subject's Signature — Date

הסכמה להשתתפות במחקר

הינך מתבקש לתת את הסכמתך להשתתפות במחקר. מיסמך זה נועד לתת לך מידע בנוגע למחקר ומהלך הבדיקות בו. כל שאלה שיש לך בנוגע למחקר אל תהסס לשאול ואנו נשתדל לענות עליה לשביעות רצונך.

שם המחקר - תגובות המו-דינאמיות למבחן כוח, ואימון במשקולות בחולי לב.

אחראי המחקר - גלילה ורבר מספר טלפון (עבודה) 09-639350'
(בית) 03-6425273

מטרת המחקר - להוסיף מידע בנוגע לתגובת הלב וכלי הדם בחולי לב לאימון במשקולות, אשר יעזור בקביעת תוכנית אימונים לחולי לב.

מהלך המחקר - מחקר זה כולל חמישה ביקורים בני שעה וחצי כל אחד אשר יערכו במחלקה למדעי החיים במכללה לחנוך גופני ולספורט ע"ש זינמן שבמכון וינגייט.

במהלך הביקור הראשון תקבל הסבר מפורט על המחקר ומהלכיו כמו כן יהיה עליך לקרוא ולחתום על טופס ההסכמה להשתתפות במחקר אשר בידיך. במהלך ביקור זה יבוצעו מדידות של גובה, משקל והערכת אחוזי שומן. כמו כן תבוצע הערכת תיפקוד הלב בעזרת רישום א.ק.ג ואקו-קארדיוגראף (צילום הלב על ידי גלי קול). הערכה תתבצע בזמן מנוחה ומיד בתום מאמץ על גבי מסילה נעה. בנוסף, מדידות לחץ דם וא.ק.ג. יבוצעו לפני, במהלך ובתום המאמץ. משך מבדק המאמץ מעורך כ-12 דקות. ביקור זה ישמש להערכת יכולתך הגופנית ובדיקה האם הינך מתאים לתנאי הקבלה למחקר.

בעת הביקור השני תתבצע בדיקת מאמץ על גבי מסילה נעה אשר תהיה דומה לזו שבוצעה במהלך הביקור הראשון. אך הפעם בזמן המיבדק נאסוף אויר אשר אתה תנשוף וכך נוכל למדוד את צריכת החמצן שלך. מדידות לחץ דם וא.ק.ג. יבוצעו לפני, במהלך ותום המאמץ. משך מבדק המאמץ מעורך כ-12 דקות.

בביקור השלישי תתבקש לבצע מבחן להערכת החוזק המירבי שלך. זאת נבצע בעזרת שני תרגילים עם משקולות. תרגיל אחד יהיה פשיטת ברכיים והתרגיל השני הינו כפיפת מרפק ביד אחת. המטרה היא למצוא את כמות המשקל המירבית שהינך יכול להרים לאורך טווח תנועה מלא בפעם אחת. תחילת המבחן יהווה חימום בו תרים משקל קל 6-8 פעמים, לאחר מכן יועלה המשקל בהתאם להרגשתך. בדרך כלל לוקח 5-6 ניסיונות להשלמת המבחן. לאורך כל ניסיון יבוצעו מדידות אקו-קארדיוגראף וא.ק.ג.. מדידות לחץ דם יבוצעו לפני ואחרי כל ניסיון.

במהלך הביקור הרביעי תתבקש לבצע את אותם שני תרגילים אך הפעם כמות המשקל תהיה באחוזים שונים (20%, 40% ו-60%) מהחוזק המירבי שמצאנו במהלך הביקור השלישי. אתה

תתבקש לבצע 10-15 חזרות בכל עומס. בין תרגיל לתרגיל תנוח כ-5 דקות. מדידות לחץ דם א.ק.ג. ואקו-קארדיוגראף יבוצעו לפני במהלך ובתום המאמץ.
הביקור החמישי יהווה חזרה של הביקור הרביעי ויארך בדיוק אותו הזמן.

סיבונים אפשריים - למבחן מאמץ מיוחס סיכון קטן להתקף לב. סך כל הסיבוכים בחולי לב הינו נמוך כ-4 מקרים לכל 10,000 בדיקות ורק מקרה מוות אחד לכל 20,000 בדיקות. הסיבוכים יכולים לכלול שינויים חריגים בלחץ הדם, אי סדירות בקצב הלב ולעיתים רחוקות מאד התקפי לב. הסיכוי להופעת אחת מהתופעות הללו תצומצם על ידי בדיקה וסינון רפואי שלהנבדקים לפני המחקר.
במהלך המחקר יבוצע מעקב מתמיד אחר לחץ הדם ורישום הא.ק.ג.. כמו כן, ציוד חירום ואנשי צוות מיומנים ובכללם רופא יהיו נוכחים במהלך הבדיקה. עליה בקצב הנשימה ותחושת עייפות בשרירים מלווה כל מאמץ גופני. תגובות אלו הינו תקינות לחלוטין ונמשכות בעת המאמץ וזמן קצר לאחריו.
מחקרים העוסקים באימון משקולות בחולי לב אשר פורסמו לאחרונה, מדווחים שלא נצפו בעיות בחולי הלב, ולמעשה שיטת אימון זו באוכלוסייה חולי הלב בטוחה. נתונים במחקרים אלו מצביעים על עליה בלחץ דם וקצב לב נמוכים בהשוואה לעליה הנצפית במאמץ אירובי. בנוסף, גם לאורך הבדיקות אשר יערכו עם המשקולות יהיו נוכחים רופא ואנשי צוות ויבקרו את מהלך הבדיקה. מקובל לחוש בכאב שרירים לאחר עבודה עם משקולות, תחושת כאב זו הינה תקינה וזמנית ותחלוף כעבור יומיים לאחר המאמץ.
הינך רשאי להורות על הפסקת המאמץ בכל רגע שתרצה, ועל הבודקים להיענות לדרישתך.

תועלת אפשרית - התועלת אשר הינך מקבל מהמחקר כוללת בדיקה מקיפה של תיפקוד הלב שלך בעת מאמץ על גבי מסילה נעה ובעת הרמת משקולות, מידע לגבי הכוח המירבי שלך בגפיים עליונות ותחתונות וכמו כן קביעת אחוזי השומן שבגופך.. בעזרת התוצאות שנקבל מהבדיקות ניתן יהיה לבסס המלצות לגבי מינון לפעילות גופנית לחולי לב במכוני שיקום לב, וכמובן לעזור לך בבניית תוכנית אימונים אישית.

הינך חופשי לא להשתתף במחקר זה. אם הינך בוחר להשתתף בו, אתה רשאי לפרוש מהמחקר בכל שלב שבו תבחר.

חתימות

הנני מסכים להשתתף אצלכם במחקר אשר יבדוק ויעריך את תיפקוד הלב ומחזור הדם שלי בעת שני סוגי מאמץ שונים, דהיינו בעת מבחן מאמץ מדורג ובעת אימון במשקולות.

אחד מאנשי צוות המחקר הסביר לי את מטרת המחקר ומהלכיו במלואם. כמו כן צוינו בפני הסיכונים והתועלת שבמחקר זה.

חתימת החוקר ________________ תאריך ________________

עד לחתימה ________________ תאריך ________________

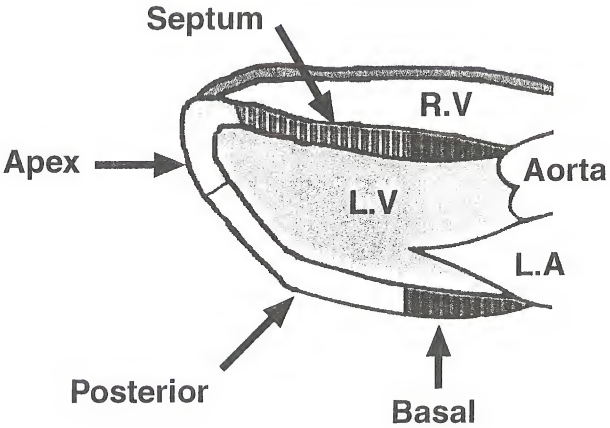
קיבלתי מידע ותיאור מדויק של המחקר המצוין לעיל כולל את מידת התועלת והסיכון בו. אני נותן בזאת את הסכמתי להשתתפות במחקר.

שם הניבדק ________________

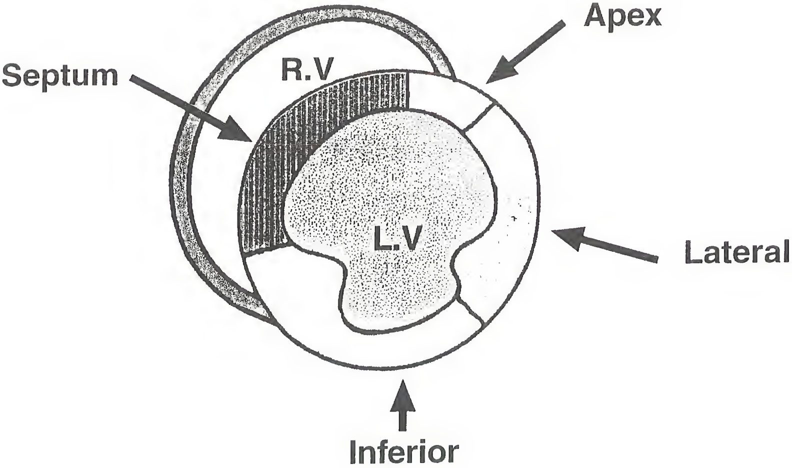
חתימת הניבדק ________________ תאריך ________________

APPENDIX C
ECHOCARDIOGRAPHIC IMAGES

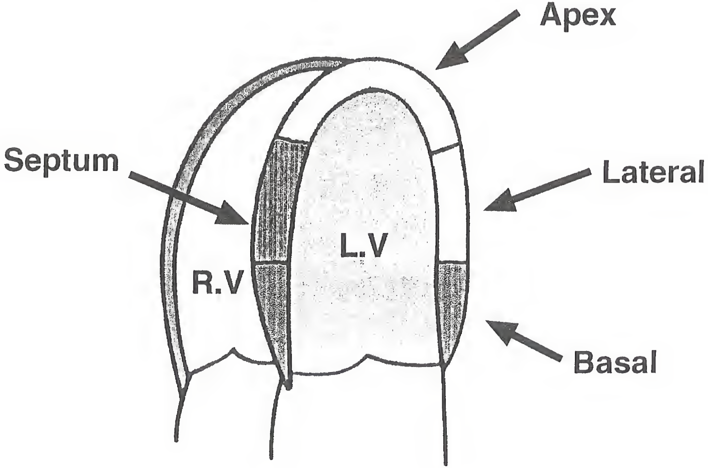
Parasternal Long Axis View



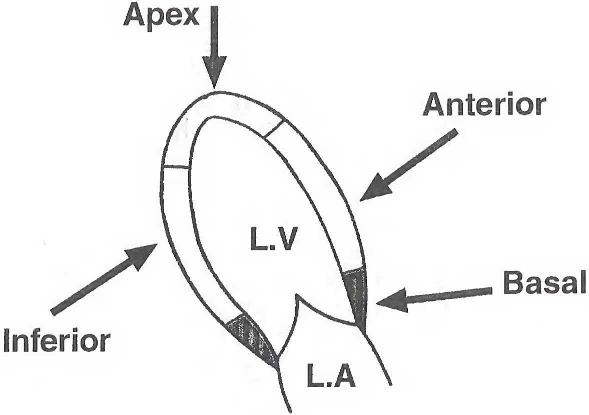
Parasternal Short Axis View



Apical 4-Chamber View



Apical 2-Chamber View



REFERENCES

Adamopoulos, S. and A.J.S. Coats. Peripheral abnormalities in chronic heart failure. Postgrad. Med. J. 67:S74-S80, 1991.

Agati, L., L. Arata, R. Luong, C. Iacoboni, M. Renzi, C.D. Vizza et al. Assessment of severity of coronary narrowing by quantitative exercise echocardiography and comparison with quantitative arteriogrphy. Am. J. Cardiol. 67:1201-1207, 1991.

Albin, G., and P.S. Ranko. Comparison of echocardiographic quantitation of left ventricular ejection fraction to radionuclide angiography in patients with regional wall motion abnormalities. Am. J. Cardiol. 65;1031-1032, 1990.

American Association of Cardiovascular and Pulmonary Rehabilitation; Guidelines for Cardiac Rehabilitation Programs; 2nd Ed; Champagne; Human Kinetics; 1995

American College of Sports Medicine; ACSM's Guidelines for Exercise Testing and Prescription; 5th Ed.; Baltimore; Williams and Wilkins, 1995.

American College of Sports Medicine. Exercise for patients with coronary artery disease. Med. Sci. Sports Exerc. 26(3):i-v, 1994.

American College of Sports Medicine. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. Med. Sci. Sports Exerc. 22:265-274, 1990.

American Heart Association Medical/Scientific Statement. Exercise Standards. A statement for health professionals from the American Heart Association. Circulation 91(2):580-615, 1995.

Asmussen, E. Similarities and dissimilarities between static and dynamic exercise. Circ. Res. 48 (Suppl. I):I3-I10, 1981.

Atkins, J.M., O.A. Matthews, J.D. Houston, G. Blomqvist, and C.B. Mullins. Arrhythmia induced by isometric (hand-grip) exercise and dynamic exercise (abstract). Clin. Res. 19:303, 1971.

Barnard R.J., R. MacAlpin, A.A. Kattus, and G.D. Buckberg. Ischemic response to sudden strenuous exercise in healthy men. Circulation 43:936-942, 1973.

Bayliss, J., M.S. Norell, R. Canepa-Anson, C. Reid, P. Poole-Wilson, and G. Sutton. Clinical importance of renin-angiotensin system in chronic heart failure double blind comparison of captopril and prazosin. Br. Med. J. 290:1861-1865, 1985.

Bertagnoli, K., P. Hanson, and A. Ward. Attenuation of exercise-induced ST depression during combined isometric and dynamic exercise in coronary artery disease. Am. J. Cardiol. 65:314-417, 1990.

Bezucha, G.R., M.C. Lenser, P.G. Hanson, and F.J. Nagle. Comparison of hemodynamic responses to static and dynamic exercise. J. Appl. Physiol 53:1589-1593, 1982.

Block, J. E., A.L. Friedlander, G.A. Brooks, P. Steiger, H. A. Stubbs, and H.K. Genant. Determinants of bone density among athletes engaged in weight-bearing and non-weight bearing activity. J. Appl. Physiol. 67(3):1100-1105, 1989.

Blomqvist, C.G., S.F. Lewis, W.F. Taylor, and R.M. Graham. Similarity of the hemodynamic responses to static and dynamic exercise of small muscle groups. Circ. Res. 48 (Suppl. I):I87-I92, 1981.

Blomqvist, C.G., and B. Saltin. Cardiovascular adaptations to physical training. Ann. Rev. Physiol. 45:169-189, 1983.

Blumenfeld, J.D., and J.H. Laragh. Congestive Heart Failure: Pathophysiology, Diagnosi and treatment; 1st Ed.; Caddo; Professional Communications Inc; 1994; pp. 1-10.

Borg, G. Subjective effort in relation to physical performance and working capacity. In Pick, H.L. Jr.(Ed.). Psychology: From Research to Practice; New York; Plenum; 1978; pp. 333-361.

Bouxsein, M.L., and R. Marcus. Overview of exercise and bone mass. Osteoporosis 20(3):787-802, 1994.

Braith , R.W., R.M. Mills, M.L. Pollock, M.A. Welsch, and J. Keller. Resistance exercise training restores bone mineral density in transplant recipients. J. Am. Coll. Cardiol. 28:1471-1477, 1996.

Braith , R.W., M.L. Pollock, R.M. Mills, M.A. Welsch, and R.L. Camberg. Rapid loss of vertebral bone mineral density after heart transplantation. Med. Sci. Sports Exerc. 26(Suppl. 5):S165, 1994.

Braith , R.W., M.C. Limacher S.H. Leggett, and M.L. Pollock. Skeletal muscle strength in heart transplant recipients. J. Heart Lung Transplant 12:1018-1023, 1993.

Braith, R.W., J.E. Graves, M.L. Pollock S.H. Leggett, and A.B. Colvin. Comparison of 2 vs 3 days/week of variable resistance training during 10- and 18- week programs. Int. J. Sports Med. 10:450-454, 1989.

Braunwald E., S.J.Sarnoff, R.B. Case, W.N. Stainsby, and G.H. Welch, JR. Hemodynamic determinants of coronary flow: effects of changes in aortic pressure and cardiac output on the relationship between myocardial oxygen consumption and coronary flow. Am. J. Physiol. 192:1570163, 1958.

Buckberg, G.D., D.E. Fixler, J.P. Archie, and J.I.E. Hofman. Experimental subendocaridal ischemia in dogs with normal coronary arteries. Cir. Res. 30:67-81, 1972.

Butler, M.R., W.H. Beierwaltes, and F.J. Rogers. The cardiovascular response to circuit weight training in patients with cardiac disease. J. Cardiopul. Rehabil. 7:402-409, 1987.

Caforio, A.L.P., B. Rossi, R. Risaliti, G. Siciliano, A. Marchetti, and C. Angelini. Type I fiber abnormalities in skeletal muscle of patients with hypertrophic and dilated cardiomyopathy: evidence of subclinical myogenic myopathy. J. Am. Coll. Cardiol. 14:1464-1473, 1989.

Certo, C.M. History of cardiac rehabilitation. Phys. Therapy. 65:1793-1795, 1985.

Chang, D., D. Slane, P. Ranko, and P. Hanson. Hemodynamic responses to leg extension exercise is determined in older cardiac patients (Abstract). Med. Sci. Sports Exerc. 26(5):S32, 1994.

Clausen, J.P. Effect of physical training on cardiovascular adjustment to exercise in man. Physiol. Rev. 57:779-815, 1977.

Codd, M.B. Epidemiology of idiopathic dilated and hypertrophic cardiomyopathy: a population-based study in Olmsted county, Minnesota, 1975-1984. Circulation 80:564-571, 1989.

Cohen, B.S. and Grant, A. Acute myocardial infarction: effect of a rehabilitation program on length of hospitalization and functional status of discharge. Arch. Phys. Med Rehabil. 54:201-206, 1973.

Cononie, C.C., J.E. Graves, and M.L. Pollock. Effect of exercise training on blood pressure in 70- to 79-year old men and women. Med. Sci. Sports Exerc. 23:505-511, 1991.

Crawford, M.H., D.H. White, and K.W. Amon. Echocardiographic evaluation of left ventricular size and performance during handgrip and upright bicycle exercise. Circulation 59:1188-1196, 1979.

Crozier-Ghilarducci, L.E., R.G. Holly, and E. A. Amsterdam. Effects of high resistance training in coronary artery disease. Am. J. Cardiol. 64:866-870, 1989.

Curtiss, C., J.N. Cohn, T. Vrobel, and J.A. Franciosa. Role of renin-angiotensin system in the systemic vasoconstriction chronic congestive heart failure. Circulation 58:763-770, 1978.

Dalen , N., and K.E. Olson. Bone mineral content and physical activity. Acta Orthop. Scand. 45:170-174, 1974.

Daub, W.D., G.P. Knapik, and W R. Black. Strength training early after myocardial infarction. J. Cardiopul. Rehabil. 16:100-108, 1996.

DeBusk, R., W. Pitts, W. Haskell, and N. Houston. Comparison of cardiovascular responses to static-dynamic effort and dynamic effort alone in patienswith chronic ischemic heart disease. Circulation 59(5):977-984, 1979.

DeBusk, R., R. Valdez, N. Houston, and W. Haskell. Cardiovascular responses to dynamic and static effort soon after myocardial infarction: application to occupational work assessment. Circulation 58(2):368-375, 1978.

Donckier, J.E., P.M. De Coster, J-L Vanoverschelde, C. Brichant, F. Cauwet, and E. Installe. Atrial natriuretic factor, cardiac volumes and filling pressures during exercise in congestive heart failure. Eur. Heart J. 12:33-337, 1991.

Drexler, H., U. Riede, T. Munzel, H. Konig, E. Funke, and H. Just. Alterations of skeletal muscle in chronic heart failure. Circulation 85:1751-1759, 1992.

Drexler, H. Reduced tolerance in chronic heart failure and its relationship to neurohumoral factors. Eur. Heart J. 12:21-28, 1991.

Drexler, H., M. Hiroi, U. Riede, U. Banhardt, T. Meinertz, and H. Just. Skeletal muscle blood flow, metabolism and morphology in chronic congestive heart failure and effects of short- and long- term angiotensin converting enzyme inhibition. Am. J. Cardiol. 62:82E-85E, 1988.

Drexler, H., U. Riede, M. Hiroi, T. Münzel, C. Holubarsch, and T. Meinertz. Ultrastructural analysis of skeletal muscle in chronic heart failure: relation to exercise capacity and indices of LV-function (Abstract). Circulation 78(Supll II):107II, 1988.

Drexler, H., F. Faude, S. Hoing, and H. Just. Blood flow distribution within skeletal muscle during exercise in the presence of chronic heart failure: effect of milrinone. Circulation 76(6):1344-1352, 1987.

Durnin, J.V.G.A., and J. Womersley. Body fat assessed from total body density and its estimation from skinfold thickness measurements on 481 men and women aged 16-72 years. Br.J. Nut. 32:77-97, 1974.

Effron, M.M. Effects of resistive training on left ventricular function. Med. Sci. Sports Exerc. 21(6):694-697, 1989.

Ehsani, A.A., W. H. Martin, G.W. Heath, and E.F. Coyle. Cardiac effects of prolonged and intense exercise training in patients with coronary artery disease. Am. J. Cardiol. 50:246-254, 1982.

Elkayam, U., A. Roth, L. Weber, W. Hsueh, M. Nanna, L. Freidenberger, A.N. Chandraratna, and S.H. Rahimtoola. Isometric exercise in patients with chronic advanced heart failure: hemodynamic and neurohumoral evaluation. Circulation 72(5):9750981, 1985.

Ewart, C.K. Psychological effects of resistive weight training: implications for cardiac patients. Med. Sci. Sports Exerc. 21(6):683-688, 1989.

Ewing, D.J., F. Kerr, R, Leggett, and A. Murray. Interaction between cardiovascular responses to sustained handgrip and Valsalva maneuver. Br. Heart J. 38:483-490, 1976.

Faigenbaum, A.D., G.S. Skrinar, W.F. Cesare, W.J. Kraemer, and H.E. Thomas. Physiologic and symptomatic response of cardiac patients to resistance exercise. Arch. Phys. Med. Rehabil. 71:395-398, 1990.

Featherstone, J. F., R.G. Holly, and E.A. Amsterdam. Physiology responses to weight lifting in coronary artery disease. Am. J. Cardiol. 71:287-292, 1993.

Feigenbaum, M.S., and M.L. Pollock. Strength training: rational for current guidelines for adult fitness programs. Phys. Sportsmed. 25(2):44-64, 1997.

Feigenbaum, H. Echocardiography; 5th Ed.; Philadelphia; Lea and Febiger; 1994; pp. 134-180.

Fiatarone M.A, E.C. Marks, N.D. Ryan, C.N. Meredith, L.A. Lipsitz, and J.W. Evans. High-intensity strength training in nonagenarians: effects on skeletal muscle. J.A.M.A. 263:3029-3034, 1990.

Fisman, E.Z., E. Ben-Ari, A. Pines, Y. Drory, M. Motro, and J.J. Kellermann. Usefulness of heavy isometric exercise echocardiography for assessing left venricualr wall motion patterns late (≥ 6 months) after acute myocardial infarction. Am. J. Cardiol. 70:1123-1128, 1992.

Fluckey, J.D., M.S. Hickey, J.K. Brambrink, K.K. Hart, K. Alexander, and B.W. Crag. Effects of resistance exercise on glucose tolerance in normal and glucose intolerant subjects. J. Appl. Physiol. 77(3):1087-1092, 1994.

Fozzard, H.A., R.B. Jennings, E. Haber, A.M. Kats, and H.E. Morgan; The Heart and Cardiovascular System Scientific Foundations. Second edition; New-York; Raven Press; 1991; pp. 333-354.

Francis, G.S. and, J.N. Cohn. Heart failure: mechanism of cardiac and vascular dysfunction and the rational for pharmacological intervention. FASEB J. 4:3068-3075, 1990.

Franklin, B.A., K. Bonzheim, S. Gordon, and G. C. Timmis. Resistance training in cardiac rehabilitation. J. Cardiopul. Rehabil. 11:99-107, 1991.

Franklin, B.A., H.K. Hellerstein, S. Gordon, and G. C. Timmis. Exercise prescription for the myocardial infarction patient. J. Cardiopul. Rehabil. 6:62-79, 1986.

Frederickson, L. Confronting mitral valve prolapse syndrome; San Marcus; Avant Books; 1988.

Froelicher, V., D. Jensen, F. Genter, M. Sullivan, M.D. McKirnan, and K Witztum. A randomized trial of exercise training in patients with coronary heart disease. J.A.M.A. 252:1291-1297, 1984.

Frontera, W.R., C.N. Meredith, K.P. O'reilly, and W.J. Evans. Strength training and determinants of VO2max in older men. J. Appl. Physiol. 68(1):329-333, 1990.

Gettman, L.R., J.J. Ayres, M.L. Pollock, and A. Jackson. The effect of circuit weight training on strength, cardiorespiratory function, and body composition of adult men. Med. Sci. Sports Exerc. 10(3):171-176, 1978.

Gettman, L.R., and M.L. Pollock. Circuit weight training a critical review of its physiological benefits. Phys. Sportsmed. 9(1):44-60, 1981.

Ginzton, L.E., M.M. Laks, M. Brizendine, R. Contant, and I. Mena. Noninvasive measurement of the rest and exercise peak systolic pressure/end systolic volume ratio: a sensitive two-dimensional echocardiographic indicator of left ventricular function. J. Am. Coll.Cardiol. 4(3)509-516, 1984.

Gobel, F.L., L A. Nordstrom, R.R. Nelson, C.R. Jorgensen, and Y. Wang. The rate-pressure product as an index of myocardial oxygen consumption during exercise in patients with angina pectoris. Circulation 57(3):549-556, 1977.

Goldberg, A.P. Aerobic and resistive exercise modify risk factors for coronary heart disease. Med. Sci. Sports Exerc. 21(6):669-674, 1989.

Goldberg, L.E., D.L. Elliot, R.W. Schutz, and F.E. Kloster. Changes in lipid and lipoprotein levels after weight training. J.A.M.A. 252:504-506, 1984.

Gordon, N.F., H.W. Kohl III, M.L. Pollock, H. Vaandrager, L.W. Gibbons, and S.N. Blair. Cardiovascular safety of maximal strength testing in healthy adults. Am. J. Cardiol. 76:851-853, 1995.

Grossman, W., E. Braunwald, T. Mann, L.P Mclaurin, and L. Green. Contractile state of the left ventricle in man as evaluated from end-systolic pressure-volume relations. Circulation 56(5):845-852, 1977.

Haissly, J-C., R. Messin, S. Degre, P. Vandermoten, B. Demart, and H. Denolin. Comparative response of isometric(static) and dynamic exercise tests in coronary disease. Am. J. Cardiol. 33:791-796, 1974.

Hamdy R. C., J.S. Anderson, K.E. Whalen, and L.M. Harvill. Regional differences in bone density of young men involved in different exercise. Med. Sci. Sports Exerc. 26(7):884-888, 1994.

Hanson, P. Exercise testing and training in patients with chronic heart failure. Med. Sci. Sports Exerc. 26(5):527-537, 1994.

Haslam, D.R.S., N. McCartney, R.S. McKelvie, and J.D. Macdougall. Direct measurements of arterial blood pressure during formal weightlifting in cardiac patients. J. Cardiopul. Rehabil. 8:213-225, 1988.

Harris, K.A., and R.G. Holly. Physiological response to circuit weight training in borderline hypertensive subjects. Med. Sci. Sports Exerc. 19:246-252, 1987.

Helela, T. Variations in thickness of cortical bone ion two populations. Ann. Clin. Res. 1:227-231, 1969.

Helfant, R.H., M.A. deVilla, and S.G. Meister. Effect of sustained isometric handgrip exercise on left ventricular performance. Circulation 44:982-993, 1971.

Hickson, R.C., M.A. Rosenkrotter, and M.M. Brown. Strength training effects on aerobic power and short-term, endurance. Med. Sci. Sports Exerc. 12(5):336-339, 1980.

Hughes, V.A., W.R. Frontera, G.E. Dallal, K.J. Lutz, E.C. Fisher, and W.J. Evans. Muscle strength and body composition: association with bone mineral density in older subjects. Med. Sci. Sports Exerc. 27(7):967-974, 1995.

Hung, J., E.P. Gordon, N. Houston, W.L. Haskell, M.L. Goris, and R.F. DeBusk. Changes in rest and exercise myocardial perfusion and left ventricular function 3 to 26 weeks after clinically uncomplicated acute myocardial infarction: effects of exercise training. Am. J. Cardiol. 54:943-950, 1984.

Hurley, B. Does strength training improve health status. Strength and Conditioning. 6:7-13, 1994.

Hurley, B.F. Effects of resistive training on lipoprotein-lipids profile: a comparison to aerobic exercise training. Med. Sci. Sports Exerc. 21(6):689-693, 1989.

Hurley, B.F., J.M. Hagberg, A.P. Goldberg, D.R. Seals, A.A. Ehsani, R.E. Brennan, and J.O Holloszly. Resistive training can reduce coronary risk factors without altering VO2max or percent body fat. Med. Sci. Sports Exerc. 20(2):150-154, 1988.

Hurley, B.F., and P.F. Kokkinos. Effects of Weight training on risk factors for coronary artery disease. Sports Med. 4:231-238, 1987.

Jackson, D.H. T.J. Reeves, L.T. Sheffield, and J. Burdeshow. Isometric effects on treadmill exercise response in healthy young men. Am. J. Cardiol. 31:344-350, 1973.

Jondeau, G., S.D. Kats, L. Zohman, M. Goldberger, M. McCarthy, J.P Bourdarias, and T.H. LeJemtel. Active skeletal muscle mass and cardiopulmonary reserve; failure to attain peak aerobic capacity during maximal bicycle exercise in patients with severe congestive heart failure. Circulation 86:1351-1356, 1992.

Just, H. Peripheral adaptations in congestive heart failure: a review. Am. J. Med. 90(Suppl 5B):23S-26S, 1991.

Kass, J.E., and R.J. Castriotta. The effect of circuit weight training on cardiovascular function in healthy sedentary males. J. Cardiopul. Rehabil. 14:378-383, 1994.

Kelemen, M.H. Resistive training safety and assessment guidelines for cardiac and coronary prone patients. Med. Sci. Sports Exerc. 21(6):675-677, 1989.

Kelemen, M.H., M.B. Effron, S.A. Valenti, and K.J. Stewart. Exercise training versus exercise training with diltiazem or propranolol: effects on maximal oxygen uptake and exercise duration in men with mild hypertension (Abstract). J. Am. Coll. Cardiol. 13:241A, 1989

Kelemen, M.H., K.J. Stewart, R.E. Gillilan, C.K. Ewart, S.A. Valenti, J.D. Manley, and MD. Kelemen. Circuit weight training in cardiac patients. J. Am. Coll. Cardiol. 7:38-42, 1986.

Kerber, R.E., R.A. Miller, and S.M. Najjar. Myocardial ischemic effects of isometric, dynamic and combined exercise in coronary artery disease. Chest 76:388-394, 1975.

Keul, J., H.-H. Dickuth, G. Simon, and M. Lehmann. Effect of static and dynamic exercise on heart volume, contractility, and left ventricular dimensions. Circ. Res. 48 (Suppl. I):I162-I170, 1981.

Kivowitz, C., W.W. Parmley, R. Donoso, H. Marcus, W. Ganz, and H.J.C. Swan. Effects of isometric exercise on cardiac performance. The grip test. Circulation 44:994-1002, 1971.

Konstam M.A., M.W. Kronenberg, J.E. Udelson, D. Kinan, J.Metherall, and N. Dolan. Effectiveness of preload reserve as a determinant of clinical status in patients with left systolic dysfunction. Am. J. Cardiol. 69:1591-1595, 1992.

Laired, W.P., D.E. Fixler, and F.D. Huffines. Cardiovascular response to isometric exercise in normal adolescents. Circulation 59:651-661, 1979.

Lentini, A.C., R.S. McKlevie, N. McCartney, C.W. Tomlinson, and J.D. MacDougall. Left ventricular response in healthy young men during heavy-intensity weight-lifting exercise. J. Appl. Physiol. 75(6):2703-2710, 1993.

Levine, T.B., G.S. Francis S.R. Goldsmith, A. Simon, and J.N. Cohn. Activity of the sympathetic nervous system and renin-angiotensin system assessed by plasma hormone levels and their relationship to hemodynamic abnormalities in congestive heart failure. Am. J. Cardiol. 49:1659-1666, 1982.

Lewis, S.F., P.G. Snell. W.F. Taylor, M. Hamra, R.M. Graham, W.A. Pettinger, and G. Blomqvist. Role of muscle mass and mode of contraction in circulatory responses to exercise. J. Appl. Physiol. 58(1):146-151, 1985.

Lewis, S.F., W.F. Taylor, R.M. Graham, W.A. Pettinger, J.E. Schutte, and G. Blomqvist. Cardiovascular responses to exercise as functions of absolute and relative work load. J. Appl. Physiol. 54(5):1314-1323, 1983.

Lipkin, D.P., A.D. Jones, J.M. Round, and P.A. Polle-Wilson. Abnormalities of skeletal muscle in patients with chronic heart failure. Int. J. of Cardiol. 18:187-195, 1988.

MacDougall, J.D., R.S. McKlevie, J.R. Moroz, D.G. Sale, N. McCartney, and F. Buick. Factors affecting blood pressure during heavy weight lifting and static contractions. J. Appl. Physiol. 73(4): 1590-1597, 1992.

MacDougall, J.D., D. Tuxen, D.G. Sale, J.R. Moroz, and J.R. Sutton. Arterial blood pressure response to heavy resistance exercise. J. Appl. Physiol. 58(3): 785-790, 1985.

Mancini, D.M., G. Walter, N. Reichek, R. Lenkinski, K.K. McCully, J.L. Mullen, and J.R. Wilson. Contribution of skeletal muscle atrophy to exercise intolerance and altered muscle metabolism in heart failure. Circulation 85:1364-1373, 1992.

Mancini, D.M., E. Coyle, A. Coggan, J. Beltz, N. Ferraro, S. Montain, and J.R. Wilson. Contribution of intrinsic skeletal muscle changes to ^{31}P-NMR skeletal muscle metabolic abnormalities in patients with chronic heart failure. Circulation 80:1338-1346, 1989.

Marcus, R. Skeletal aging understanding the functional and structural basis of osteoporosis. Trends Endocrinol. Metab. 2:53-58, 1991.

Massie, B.M., M. Conway, B. Rajagopala, R. Yonge, S. Frostick, and J. Ledingham. Skeletal muscle metabolism during exercise under ischemic conditions in congestive heart failure evidence for abnormalities unrelated to blood flow. Circulation 78:320-326, 1988.

Massie, B.M., M. Conway, R. Yonge, S. Frostick, J. Ledingham, and P. Sleight. Skeletal muscle metabolism in patients with congestive heart failure: relation to clinical severity and blood flow. Circulation 1009-1019, 1987.

Maurer, G., and N.C. Nanda. Two dimensional echocardiographic evaluation of exercise-induced left and right ventricular asynergy: correlation with thallium scanning. Am. J. Cardiol. 48:720-727, 1981.

McCartney, N., R.S. McKlevie, D.R. Haslam, and N. L. Jones. Usefulness of weightlifting training in improving strength and maximal power output in coronary artery disease. Am. J. Cardiol. 67:939-945, 1991.

McCartney, N., N.B. Oldridge, A. Hicks, and N.L. Jones. Maximal isokinetic cycle ergometer in patients with coronary artery disease. Med. Sci. Sports Exerc. 21(3):313-318, 1989

McKelvie, R.S. N. McCartney, C. Tomlinson, R. Bauer, and J.D. MacDougall. Comparison of hemodynamic responses to cycling and resistance exercise in congestive heart failure secondary to ischemic cardiomyopathy. Am. J. Cardiol. 76:977-979, 1995.

McKelvie, R.S. and N. McCartney. Weightlifting in cardiac patients considerations. Sports Medicine. 10(6):355-364, 1990.

Menkes, A., S. Mazel, R.A. Redmond, K. Koffler, C. R. Libanati, C.M. Gundberg, T.M. Zizic, J. M. Hagberg, R.E. Pratley, and B.F. Hurley. Strength training increases regional bone mineral density and bone remolding in middle-age and older men. J. Appl. Physiol. 74(5):2478-2484, 1993.

Messier, S.P. and M.E. Dill. Alterations in strength and maximal oxygen uptake consequent to nuatilus circuit weight training. Res. Q. Exerc. Sport 56(4):345-351, 1985.

Miles, D.S., J.J. Owens, J.C. Golden, and R.W. Gotshall. Central and peripheral hemodynamics during maximal leg extension exercise. Eur. J. Appl. Phyiol. 56:12-17, 1987.

Miller JP, Pratley RE, and Goldberg AP. Strength training increases insulin action in healthy 50-to 65-yr-old men. J. Appl. Physiol. 77(3):1122-1127, 1994.

Misner, J.E., S.B. Going, B.H. Massey, T.E. Ball, M.G. Bemben, and L. K. Essandoh. Cardiovascular response to sustained maximal voluntary static muscle contraction. Med. Sci. Sports Exerc. 22(2):194-199, 1990.

Mitchell, J.H., F.C. Payne, B. Saltin, and B. Schibye. The role of muscle mass in the cardiovascular response to static contractions. J. Physiol. London. 309:45-54, 1980.

Mullins, C.B., and G. Blomqvist. Isometric exercise and cardiac patient. Texas Med. 69:53-58, 1973.

Munnings, F. Strength training- not for young. Physician Sportsmed. 21:133-40, 1993.

Musch, T.I., and J.A Terrell. Skeletal muscle blood flow abnormalities in rats with a chronic myocardial infarction: rest and exercise. Am. J. Physiol. 262(31):H411-H419, 1992.

Nelson, R.R., F.L. Gobel, C.R. Jorgenson, K. Wang, Y. Wank, and H.L. Taylor. Hemodynamic predictors of myocardial oxygen consumption during static and dynamic exercise. Circulation 50:1179-1189, 1974

Oldrige, N.B., N. McCartney, A. Hicks and N.L. Jones. Improvement in maximal isokinetic cycle ergometer with cardiac rehabilitation. Med. Sci. Sports Exerc. 21(3):308-312, 1989.

Painter P., and P. Hanson. Isometric exercise: implications for the cardiac patient. Cardiovasc. Rev. Rep. 5:261-279, 1984.

Partely, R., B. Nickolas, M. Rubin, J. Miller, A. Smith, M. Smith, B. Hurely, and A. Goldberg. Strength training increases resting metabolic rate and norepinephrine levels in healthy 50-to 65-yr-old men. J. Appl. Physiol. 76(1):133-137, 1994.

Pepine C.J., and W.W. Nichols. Effects of transient increase in intrathoracic pressure on hemodynamic determinants of myocardial oxygen supply and demand. Clin. Cardiol. 11:831-837, 1988.

Pepine, C.J., and L. Wiener. Effects of the Valsalva maneuver on myocardial ischemia in patients with coronary artery disease. Circulation 59:1304-1311, 1979.

Perez-Gonzale, J. F. Factors determining the blood pressure responses to isometric exercise. Circ. Res. 48 (Suppl. I):I70-I76, 1981.

Perez-Gonzale, J. F., N.R. Schiller, and W.W. Parmley. Direct and noninvasive evaluation of the cardiovascular response to isometric exercise. Circ. Res. 48 (Suppl. I):I138-I149, 1981.

Pierre-Yves, M., J.M. Escanye, F. Brunotte, B. Robin, P. Walker, and F. Zannad. Skeletal muscle metabolism in the leg during exercise in patients with congestive heart failure. Clin. Sc. 78:515-519, 1990.

Pollock, M.L., J.F. Carroll, J. E.. Graves, S. H. Leggett, R.W. Braith, M. Limacher, and J.M. Hagberg. Injuries and adherence to walk/jog and resistance training. Med. Sci. Sports Exerc. 23(10):1194-1200, 1991.

Pollock, M.L., and J.H. Wilmore; Exercise in Health and Disease: Evaluation and Prescription for Prevention and Rehabilitation; 2nd Ed; Philadelphia; W.B. Saunders Company; 1990; pp. 3-47,228.

Rajagopalan, B., M.A. Conway, B. Massie, and G. Radda. Alterations of skeletal muscle metabolism in humans studied by phosphorus 31 magnetic resonance spectroscopy in congestive heart failure. Am. J. Cardiol. 62:53E-57E, 1988.

Reichek, N., J. Wilson, M. Sutton, T. Plappert, S. Goldberg, and J. Hirshfeld. Noninvasive determination of left ventricular end-systolic stress: validation of the method and initial application. Circulation 65:99-108, 1982.

Robertson, W.S., H. Feigenbaum, W.F. Armstrong, J.C. Dillon, J. O'Donnel, and P.W. McHenry. Exercise echocardiograpy: a clinically practical addition in the evaluation of coronary artery disease. J. Am. Coll. Cardiol. 2:1085-1091, 1983.

Reddy, H.K. , K.T. Weber, J.S. Janicki, and P.A. McElroy. Hemodynamic, ventilatory and metabolic effects of light isometric exercise in patients with chronic heart failure. J. Am. Coll. Cardiol. 12:353-358, 1988.

Ryden, L. When and how to use angiotensin-converting enzyme inhibition in congestive heart failure. Am. J. Cardiol. 62:75A-80A, 1988.

Sabbah, H.N., F. Hansen-Smith, V.G. Sharov, T. Kono, M. Lesch, and P.J. Gengo. Decreased proportion of type I myofibers in skeletal muscle of dogs with chronic heart failure. Circulation 87:1729-1737, 1993.

Sagawa, K., H. Suga, A. Shouka, and K. Bakalar. End-systolic pressure/volume ratio: a new index of ventricular contractility. Am. J. Cardiol. 40:748-753, 1977.

Sagiv, M., P.G. Hanson, D. Ben-Sira, and F. Nagle. Direct vs indirect blood pressure at rest and during isometric exercise in normal subjects. Int. Sports Med. 16:514-518, 1995.

Sagiv, M., P. Hanson, M. Besozzi, F. Nagle, and L. Zager. Left ventricular responses to upright isometric handgrip and deadlift in men with coronary artery disease. Am. J. Cardiol. 55:1296-1302, 1985

Saldivar, M., W.M. Frye, C.M. Pratt, and J.A. Herd. Safety of a low weight, low repetitions strength training program (STP) in patients with heart disease (HD) (Abstract). Med. Sci. Sports Exerc. 15:119, 1983.

Seals, D.R., R.A. Washburn, P.G. Hanson, P.L. Painter, and F.J. Nagle. Increased cardiovascular response to static contraction of larger muscle groups. J. Appl. Pysiol. 54(2):434-437, 1983.

Shaw, C.E., K.K. McCully, and J.D. Posner. Injuries during the one repetition maximum assessment in the elderly. J. Coardiopul. Rehabil. 15:283-283, 1995.

Sheilkh, K.H., J.R. Bengtson, S. Helmy, C. Juarez, R. Burgress, T.M. Bashore, and J. Kisslo. Relation of quantitative coronary lesion measurements to the development of exercise-induced ischemia assessed by exercise echocardiography. J. Am. Cardiol. 15:1043-1051, 1990.

Shephard, R.J. The value of exercise in ischemic heart disease: accumulative analysis. J. Cardiac. Rehab. 3:294-298, 1983.

Siri, W.E. Body composition from fluid spaces and density. In Brozek, J., and A. Henschel (Eds); Techniques for measuring body composition; Washington DC.; National Academy of Science; 1961; pp. 223-244.

Smith J.J., and J.P. Kampine; Circulatory Physiology - The Essential; Baltimore: Williams and Wilkins; 1990; pp. 16-30, 260.

Smith, R.F., G. Johnson, S, Zieche, G, Bhat, K. Blankenship, and J.N. Cohn. Functional capacity in heart failure: comparison of methods for assessment and their relation to other indexes of heart failure. Circulation 87(Suppl. VI):VI-88VI93, 1993.

Smutok, M.A., C. Reece, P.F. Kokkinos, C. Farmer, P Dawson, R. Shulman, J. DeVane-Bell, J. Patterson, C. Charabogos, A.P. Goldberg, and B. F. Hurley. Aerobic versus strength training for risk factor intervention in middle-aged men at high risk for coronary heart disease. Metabolism 42(2):177-184, 1993.

Sparling, P.B., J.D. Cantwell, C.M. Dolan, and R.K. Neiderman. Strength training in cardiac rehabilitation program: a six month follow up. Arch. Phys. Med. Rehabil. 71:148-152, 1990.

Sparling P.B., and J.D Cantwell. Strength training guidelines for cardiac patients. The Phys Sportsmedicine. 17(3):190-196, 1989

Squires, R.W., A.J. Muri, L.J. Anderson, T.G. Allison, T.D. Miller, and G.T. Gau. Weight training during phase III (early outpatient) cardiac rehabilitation: heart rate and blood pressure responses. J. Cardiopul. Rehabil. 11:360-364, 1991.

Starkey, D.B., M.L. Pollock, Y. Ishida, M.A. Welch, W.F. Brechue, J.E. Graves, and M. S. Feigenbaum. Effect of resistance training volume on strength and muscle thickness. Med. Sci. Sports Exerc. 28(10):1311-1320, 1996.

Stefadouros M.A., W. Grossman, M.E. El Shahawy, and M. Whitman. The effect of isometric exercise on left ventricular volume in normal man. Circulatrion 49:1185-1189, 1974.

Stewart, K.J., L.D. McFarland, J.J. Weinhofer, C.S. Brown, and E. P. Shapiro. No evidence for adverse clinical responses with weight training after acute myocardial infarction (Abstract). Med. Sci. Sports Exerc. 27(5):S32, 1995.

Stewart, K.J. Resistive training effects on strength and cardiovascular endurance in cardiac and coronary prone patients. Med. Sci. Sports Exerc. 21(6):678-682, 1989.

Stewart, K.J., M.B. Effron, S.A. Valenti, and M.H. Kelemen. Circuit weight training with diltiazem or propranolol: effects on muscle strength in men with mild hypertension(Abstract). Med. Sci. Sports Exerc. 21:S107, 1989.

Stewart, K.J., M. Mason, and M.H. Kelemen. Three-year participation in circuit weight training improves muscular strength and self-efficacy in cardiac patients. J. Cardiopul. Rehabil. 8:292-296, 1988.

Stone, M.H., S.J. Fleck, N.T. Triplett, and W.J. Kreamer. Health- and performance-related potential of resistance training. Sports Med. 11(4):210-231, 1991.

Stone, M.H. Implications for connective tissue and bone alterations resulting from resistance exercise training. Med. Sci. Sports Exerc. 20(5)-S162-S169, 1988.

Stowers, T, J. McMillan, D. Scala, V. Davis, D. Wilson, and M. Stone. The short-term effects of three different strength-power training methods. Natl. Strength Condit. Assoc. J. 5:24-27, 1983.

Stralow, C.R., T.E. Ball, and M. Looney. Acute cardiovascular responses of patients with coronary disease to dynamic variable resistance exercise of different intensities. J. Cardiopul. Rehabil. 13:255-263, 1993.

Stratton, J.R., J.F. Dunn, S. Adamopoulos, G.J. Kemp, A.J.S. Coats, and B, Rajagopalan. Training partially reverses skeletal muscle metabolic abnormalities during exercise in heart failure during exercise in heart failure. J. Appl. Physiol. 76(4):1575-1582, 1994.

Sullivan, M.J., M.B. Higginbotham, and F.R. Cobb. Exercise training in patients with chronic heart failure delays ventilatory anaerobic threshold and improves submaximal performance. Circulation 79;324-329, 1989.

Takeuchi, M., Y. Igarashi, S. Tomomoto, M. Odake, T. Hayashi, and T. Tsukamoto. Single-beat estimation of the slope at the end-systolic pressure-volume relation in the human left ventricle. Circulation 83:202-212, 1991.

Tesch, P.A. Skeletal muscle adaptations consequent to long-term resistance exercise. Med. Sci. Sports Exerc. 20(5):S132-S134, 1988.

Treasure, C.B., and R.W. Alexander. The dysfunctional endothelium in heart failure. J. Am. Coll. Cardiol. 22:129A-143A, 1993.

Ulrich, I. H., C.M. Reid, and R.A. Yeater. Increased HDL-cholesterol levels with a weight training program. South Med. J. 80:328-331, 1987.

Vander, L.B., B.A. Franklin, D. Wrisley, and M. Rubenfire. Acute cardiovascular responses to Nautilus exercise in cardiac patients: implications for exercise training. Ann. Sports Med. 2:165-169, 1986.

Vatner, S.F., and L. Huttinger. Coronary vascular mechanisms involved in decompensation from hypertrophy to heart failure. J. Am. Coll. Cardiol. 22:34A-40A, 1993.

Verrill, D.E., and P.M. Ribisl. Resistive exercise training in cardiac rehabilitation: an update. Sports Medicine. 21(5):347-383, 1996.

Verrill, D., E. Shoup, G. McElveen, K. Witt and D. Bergey. Resistive exercise training in cardiac patients. Sports Med. 13(3):171-193, 1992.

Weber, K.T., J.S. Janicki, C. Campbell, and R. Replogle. Pathophysiology of acute and chronic heart failure. Am. J. Cardiol. 60:3C-6C, 1985.

Wiecek, E.M., N. McCartney, and R.S. McKelvie. Comparison of direct and indirect measures of systemic arterial pressure during weightlifting in coronary artery disease. Am. J. Cardiol. 66:10655-1069, 1990.

Wiener, D.H., L.I. Fink, J. Maris, R.A. Jones, B. Change, and J.R. Wilson. Abnormal skeletal muscle bioenergetics during exercise in patients with heart failure: role of reduced muscle blood flow. Circulation 73(6):1127-1136, 1986.

Wilmore J.H. The aging bone and muscle. Sports Med 10(2):231-244, 1991.

Wilmore, J.H., R.B. Parr, and R.N. Girandola. Physiological alterations consequent to circuit weight training. Med. Sci. Sports Exerc. 10:79-84,1978.

Wilmore, J., R. Parr, P. Vodak, T. Barstow, T. Pipes, P. Ward, and P. Leslie. Strength, endurance, BMR, and body composition changes with circuit weight training (Abstract). Med. Sci. Sports Exerc. 8:59-60,1976.

Wilson, J.R., L. Fink, J. Maris, N. Ferraro, J. Power-Vanwart, S. Eleff, and B. Change. Evaluation of energy metabolism in skeletal muscle of patients with heart failure with gated phosphorus-31 nuclear magnetic resonance. Circulation 71(1):57-62, 1985.

Wilson, J.R., Martin, J.L, D. Schwartz, and N. Ferraro. Exercise intolerance in patients with chronic heart failure: role of impaired nutritive flow to skeletal muscle. Circulation 69:1079-1087, 1984.

Yancy, C.W., D. Parsons, L. Lane, M. Carry, B.G. Firth, and C.G. Blomqvist. Capillary density, fiber type, and enzyme composition of skeletal muscle in congestive heart failure (Abstract). J. Am. Coll. Cardiol. 13(Suppl. A):38A, 1989.

Zelis, R., L.I. Sinoway, T.I. Musch, D. Davis, and H. Just. Regional blood flow in congestive heart failure; concept of compensatory mechanism with short and long time constant. Am. J. Cardiol. 62:2E-8E, 1988.

BIOGRAPHIC SKETCH

Galila Werber served in the Israeli defense force as a sport instructor between the years 1981-1983. Ms. Werber received her Bachelor of Education in physical education from the Zinman College at the Wingate Institute, Israel in 1987. Upon graduation, she worked at the Tihon Hadash Junior-High school in Tel Aviv, Israel, as a physical education teacher during the years 1987 to 1989. Concurrently, she enrolled in the graduate program at Tel-Aviv University, Israel, and graduated with a Master of Science in exercise physiology in 1991. Along with her graduate studies, Ms. Werber worked in the Life Sciences and Sports Medicine department at Zinman College in which she administrated the cardiac rehabilitation program. A strong interest in exercise physiology led to the pursuit of a doctoral degree at the University of Florida in Gainesville, Florida. During this time, Ms. Werber was employed as a graduate teaching and research assistant. Presently Ms. Werber is employed as the Head of Cardio-Pulmonary Rehabilitation Studies in the Zinman College at the Wingate Institute and as a consultant in exercise physiology at the Procardia-Cardiostyle cardiac rehabilitation center in Tel Aviv, Israel.

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Michael L. Pollock, Chairman
Professor of Exercise and Sport Sciences

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Randy Braith

Randy W. Braith
Assistant Professor of Exercise and Sport Sciences

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Philip Posner

Philip Posner
Professor of Physiology

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

David T, Lowenthal
Professor of Exercise and Sport Sciences

This dissertation was submitted to the Graduate Faculty of the College of Health and Human Performance and to the Graduate School and was accepted as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

December, 1997

Dean, College of Health and
Human Performance

Dean, Graduate School